



- Cardiology (290)
- Clinical haematology/oncology (171)
- Clinical pharmacology and toxicology (273)
- Clinical sciences (410)
- Dermatology (131)
- Endocrinology (174)
- Gastroenterology (183)
- Infectious diseases and STIs (212)
- Nephrology (104)
- Neurology (247)
- Ophthalmology (56)
- Psychiatry (59)
- Respiratory medicine (162)
- Rheumatology (134)

2600 MCQ with Illustrated Answer
Full Topics Note
14 Chapter

14 Chapter

1

Our

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P. M MCQ Bank 2017



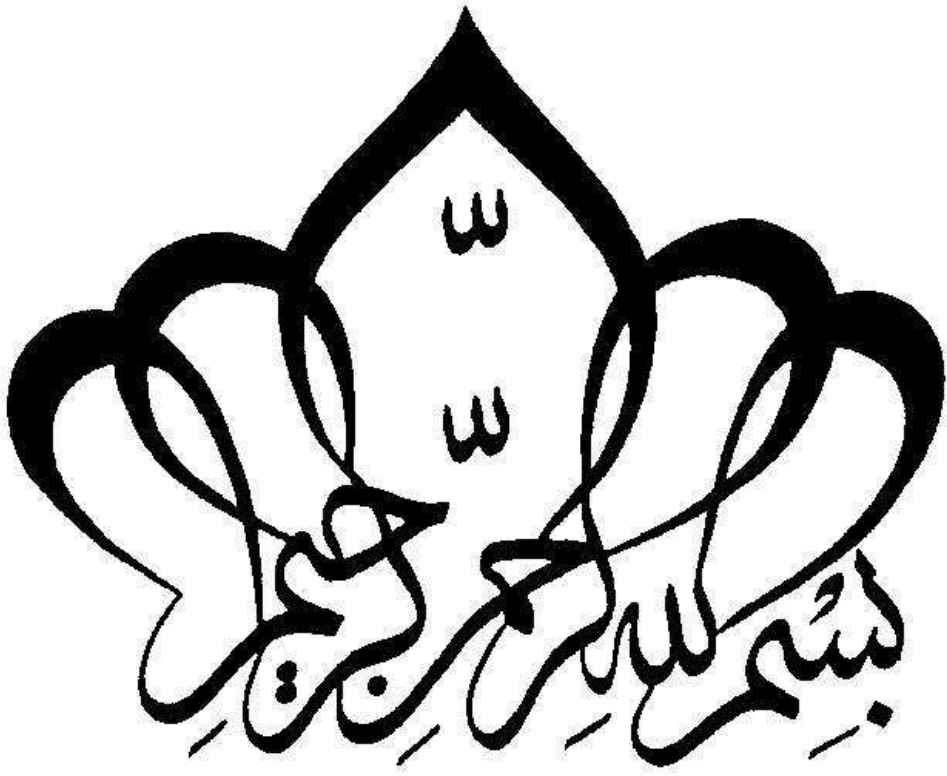
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P. M MCQ Bank 2017



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قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
۞ إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

إهداء

للكل آية من كتاب الله حفظناها ولكل معلومة قرأناها ونسيناها لعل الله يحمينا من النسيان ويروها إلينا
للكل شخص وعونا له في ظهر الغيب لعل الله أن يستجيب دعائنا
للكل مريض قرأ له ما أله لعل الله يشفيه ويعير إليه صحته وعافيته
للكل لحظة تألم بها أحببنا لعل الله لا يريهم الألم ولا يضييهم مرة أخرى
للكل موقف اجتجت به عيناى فلم تلبيانى لعل الله لا يضرنى بهما في حياتى
للكل حالم صاحب أهراف ومباوى لعل الله يحقق له مقاصده حتى يرضيه ويرضى عنه
للكل مجتهد لعل الله يجعل له من التوفيق أوفر حظ ونصيب ويهون له الأسباب كلها من حيث لا يدري من غير ضراء مضرة ولا فتنة مضلة
للكل من فقر شيئا في الحياة لعل الله يعيره إليه أو يعوضه بما يسعده
للكل شخص ابتعدنا عنه لعل الله يجمعه بمن هم أفضل منا
وأخيرا وليس آخرا لأهلنا لعل الله يجعلها لهم صرقة جارية ترو إليهم قليلا من فضلهم علينا وتكون سببا يلم شملنا تحت سقف واحد بعد أن
هجرتنا الحروب والمصائب

يقول أحد الصالحين

إِذَا رَأَيْتَ أَنَّ اللَّهَ وَفَقَكَ لِإِخْوَةِ تَعْيْنِكَ عَلَى الْخَيْرِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ بِكَ خَيْرًا !! وَإِذَا وَفَقَكَ اللَّهَ لِلدُّعَاءِ.. فَأَعْلَمْ أَنَّهُ يَرِيدُ أَنْ يُعْطِيَكَ !! وَإِذَا
وَفَقَكَ لِقَضَاءِ حَوَائِجِ النَّاسِ فَأَعْلَمْ بِأَنَّهُ لَنْ يَتَخَلَّى عَنْكَ !! وَإِذَا وَفَقَكَ اللَّهَ لِذِكْرِهِ.. فَأَعْلَمْ أَنَّهُ يُحِبُّكَ !!.. وَإِذَا أُحِبَّكَ أَحَدٌ.. وَنَصَرَكَ.. وَأَيَّرَكَ..
وَأُسْتَجَابَ لِدُعَائِكَ !!

This issue of **MRCPPART1 MADE EASY 2017** contains the following:

- Full topic note.
- Around 2600 MCQs from **PM 2017 BANK** covering 14 chapter in internal medicine
- External links for the related guidelines (such as NICE, SIGN, RCP ...etc).
- Comments with illustrative pictures and links to external media.
- Most Recommended QBank for MRCP Part 1, Arab Board, Jordanian Board and other internal medicine assessment exams.

Please Note that

Each chapter begins with notes section. These notes are repeated under the Questions in the website. In these collections the notes are not repeated under each question except in some to avoid unnecessary repetition. For clarity and convenience the title of the related note is only mentioned after the comments in case the reader want to refer back to the respective note in the notes section.

The illustration below is to show and Example

☐	Minimal change disease
☐	Post-streptococcal glomerulonephritis
☐	Goodpasture's syndrome

Goodpasture's syndrome

- IgG deposits on renal biopsy
- anti-GBM antibodies

• These changes are characteristic of Goodpasture's syndrome.

[Discuss and give feedback](#)

Goodpasture's syndrome 

Comment of the Q

title of the note in case the reader want to refer back to the notes sections

MRCPPART1 MADE EASY 2017

Chapter	Number of Q
Cardiology	292
Clinical haematology/oncology	171
Endocrinology	174
Gastroenterology	183
Infectious diseases and STIs	212
Nephrology	104
Neurology	247
Respiratory medicine	162
Rheumatology	134
Clinical pharmacology and toxicology	273
Clinical sciences	410
Dermatology	131
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Psychiatry	59

إعداد وتنسيق

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&

A. MURAD

لا تنسونا من صالح دعائكم

ما كان من خطأ فمن أنفسنا وما كان من توفيق فمن الله

تم بحمد الله وتوفيقه ومنه

جعل الله عملنا متقبلاً خالصاً لوجهه الكريم

Reference ranges

Reference ranges vary according to individual labs. All values are for adults unless otherwise stated

Full blood count

Haemoglobin	Men: 13.5-18 g/dl Women: 11.5-16 g/dl
Mean cell volume	82-100 fl
Platelets	150-400 * 10 ⁹ /l
White blood cells	4-11 * 10 ⁹ /l

Urea and electrolytes

Sodium	135-145 mmol/l
Potassium	3.5 - 5.0 mmol/l
Urea	2.0-7 mmol/l
Creatinine	55-120 umol/l
Bicarbonate	22-28 mmol/l
Chloride	95-105 mmol/l

Liver function tests

Bilirubin	3-17 umol/l
Alanine transferase (ALT)	3-40 iu/l
Aspartate transaminase (AST)	3-30 iu/l
Alkaline phosphatase (ALP)	30-100 umol/l
Gamma glutamyl transferase (yGT)	8-60 u/l
Albumin	35-50 g/l
Total protein	60-80 g/l

Other haematology

Erythrocyte sedimentation rate (ESR)	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Prothrombin time (PT)	10-14 secs
Activated partial thromboplastin time (APTT)	25-35 secs
Ferritin	20-230 ng/ml
Vitamin B12	200-900 ng/l
Folate	3.0 nmol/l
Reticulocytes	0.5-1.5%
D-Dimer	< 400 ng/ml

Other biochemistry

Calcium	2.1-2.6 mmol/l
Phosphate	0.8-1.4 mmol/l
CRP	< 10 mg/l
Thyroid stimulating hormone (TSH)	0.5-5.5 mu/l
Free thyroxine (T4)	9-18 pmol/l
Total thyroxine (T4)	70-140 nmol/l
Amylase	70-300 u/l

Uric acid	0.18-0.48 mmol/l
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Arterial blood gases

pH	7.35 - 7.45
pCO2	4.5 - 6.0 kPa
pO2	10 - 14 kPa

Lipids

Desirable lipid values depend on other risk factors for cardiovascular disease, below is just a guide:

Total cholesterol	< 5 mmol/l
Triglycerides	< 2 mmol/l
HDL cholesterol	> 1 mmol/l
LDL cholesterol	< 3 mmol/l

Acute epiglottitis	Notes
Animal bites	Notes
Anthrax	Notes
Antibiotics: anaerobic activity	Notes
Antibiotics: bactericidal vs. bacteriostatic	Notes
Antifungal agents	Notes
Bacterial vaginosis	Notes
Brucellosis	Notes
Campylobacter	Notes
Cat scratch disease	Notes
Cellulitis	Notes
Chickenpox	Notes
Chickenpox exposure in pregnancy	Notes
Chikungunya	Notes
<i>Chlamydia</i>	Notes
Cholera	Notes
Classification of bacteria	Notes
Congenital infections	Notes
Dengue fever	Notes
DNA viruses	Notes
Epididymo-orchitis	Notes
Epstein-Barr virus: associated conditions	Notes
<i>Escherichia coli</i>	Notes
Gastroenteritis	Notes
Genital warts	Notes
Giardiasis	Notes
Gonorrhoea	Notes

H1N1 influenza pandemic	Notes
Hand, foot and mouth disease	Notes
Hepatitis B	Notes
Hepatitis B and pregnancy	Notes
Hepatitis B serology	Notes
Hepatitis E	Notes
HIV and pregnancy	Notes
HIV: diarrhoea	Notes
HIV: immunisation	Notes
HIV: Kaposi's sarcoma	Notes
HIV: Mycobacterium avium complex	Notes
HIV: neurocomplications	Notes
HIV: opportunistic infections and other disorders	Notes
HIV: <i>Pneumocystis jiroveci</i> pneumonia	Notes
HIV: seroconversion	Notes
Identifying gram-positive bacteria	Notes
Incubation periods	Notes
Infectious mononucleosis	Notes
Infective endocarditis: prognosis and management	Notes
Leishmaniasis	Notes
Leprosy	Notes
Leptospirosis	Notes
Linezolid	Notes
Listeria	Notes
Lyme disease	Notes
Lyme disease: features	Notes
Lymphadenopathy	Notes

Malaria	Notes
Malaria: Falciparum	Notes
Malaria: non-falciparum	Notes
Malaria: prophylaxis	Notes
Measles	Notes
Meningitis: causes	Notes
Meningitis: CSF analysis	Notes
Meningitis: management	Notes
Meningococcal septicaemia: investigations	Notes
MRSA	Notes
Necrotising fasciitis	Notes
Nematodes	Notes
Orf	Notes
Osteomyelitis	Notes
Pelvic inflammatory disease	Notes
Post-exposure prophylaxis	Notes
Pyogenic liver abscess	Notes
Pyrexia of unknown origin	Notes
Rabies	Notes
Rickettsiae	Notes
RNA viruses	Notes
<i>Salmonella</i>	Notes
Scabies	Notes
Schistosomiasis	Notes
<i>Shigella</i>	Notes
Splenectomy	Notes
Staphylococcal toxic shock syndrome	Notes

Chapter: Infectious Diseases & STI

Staphylococci	Notes
STI: ulcers	Notes
Streptococci	Notes
<i>Strongyloides stercoralis</i>	Notes
Syphilis	Notes
Syphilis: investigation	Notes
Syphilis: management	Notes
Tape worms	Notes
Tetanus	Notes
Tetanus: vaccination	Notes
Toxoplasmosis	Notes
Trypanosomiasis	Notes
Tuberculosis: diagnosis	Notes
Urinary tract infection in adults: management	Notes
Vaccinations	Notes
Vaginal discharge	Notes
Vancomycin	Notes
Virulence factors	Notes
Zika virus	Notes

Acute epiglottitis

Acute epiglottitis is rare but serious infection caused by *Haemophilus influenzae* type B. Prompt recognition and treatment is essential as airway obstruction may develop. Epiglottitis was generally considered a disease of childhood but in the UK it is now more common in adults due to the immunisation programme. The incidence of epiglottitis has decreased since the introduction of the Hib vaccine

Features

- rapid onset
- high temperature, generally unwell
- stridor
- drooling of saliva

Animal bites

The majority of bites seen in everyday practice involve dogs and cats. These are generally polymicrobial but the most common isolated organism is *Pasteurella multocida*.

Management

- cleanse wound
- current BNF recommendation is co-amoxiclav
- if penicillin-allergic then doxycycline + metronidazole is recommended

External Links

[Clinical Microbiology Reviews](#)

Microbiology of Animal Bite Wound Infections

Anthrax

Anthrax is caused by *Bacillus anthracis*, a Gram positive rod. It is spread by infected carcasses. It is also known as Woollsorters' disease. *Bacillus anthracis* produces a tripartite protein toxin.

- protective antigen
- oedema factor: a bacterial adenylate cyclase which increases cAMP
- lethal factor: toxic to macrophages

Features

- causes painless black eschar (cutaneous 'malignant pustule', but no pus)
- typically painless and non-tender
- may cause marked oedema
- anthrax can cause gastrointestinal bleeding

Management

- the current Health Protection Agency advice for the initial management of cutaneous anthrax is ciprofloxacin
- further treatment is based on microbiological investigations and expert advice

External Links

[Health Protection Scotland](#)

Anthrax FAQs

Antibiotics: anaerobic activity

The following antibiotics have anti-anaerobic activity:

- penicillins
- cephalosporins (except ceftazidime)
- erythromycin
- metronidazole
- tetracycline.

The following antibiotics do not have anti-anaerobic activity

- gentamicin
 - ciprofloxacin
 - ceftazidime
-

Antibiotics: bactericidal vs. bacteriostatic

Bactericidal antibiotics:

- penicillins
- cephalosporins
- aminoglycosides
- nitrofurantoin
- metronidazole
- quinolones
- rifampicin
- isoniazid.

Bacteriostatic antibiotics:

- chloramphenicol
 - macrolides
 - tetracyclines
 - sulphonamides
 - trimethoprim
-

Antifungal agents

Drug	Mechanism of action	Adverse effects	Notes
Azoles	Inhibits 14 α -demethylase which produces ergosterol	P450 inhibition Liver toxicity	
Amphotericin B	Binds with ergosterol forming a transmembrane channel that leads to monovalent ion (K ⁺ , Na ⁺ , H ⁺ and Cl ⁻) leakage	Nephrotoxicity, flu-like symptoms, hypokalaemia, hypomagnasaemia	Used for systemic fungal infections
Terbinafine	Inhibits squalene epoxidase		Commonly used in oral form to treat fungal nail infections
Griseofulvin	Interacts with microtubules to disrupt mitotic spindle	Induces P450 system, teratogenic	
Flucytosine	Converted by cytosine deaminase to 5-fluorouracil, which inhibits thymidylate synthase and disrupts fungal protein synthesis	Vomiting	
Caspofungin	Inhibits synthesis of beta-glucan, a major fungal cell wall component	Flushing	
Nystatin	Binds with ergosterol forming a transmembrane channel that leads to monovalent ion (K ⁺ , Na ⁺ , H ⁺ and Cl ⁻) leakage		As very toxic can only be used topically (e.g. for oral thrush)

Bacterial vaginosis

Bacterial vaginosis (BV) describes an overgrowth of predominately anaerobic organisms such as *Gardnerella vaginalis*. This leads to a consequent fall in lactic acid producing aerobic lactobacilli resulting in a raised vaginal pH.

Whilst BV is not a sexually transmitted infection it is seen almost exclusively in sexually active women.

Features

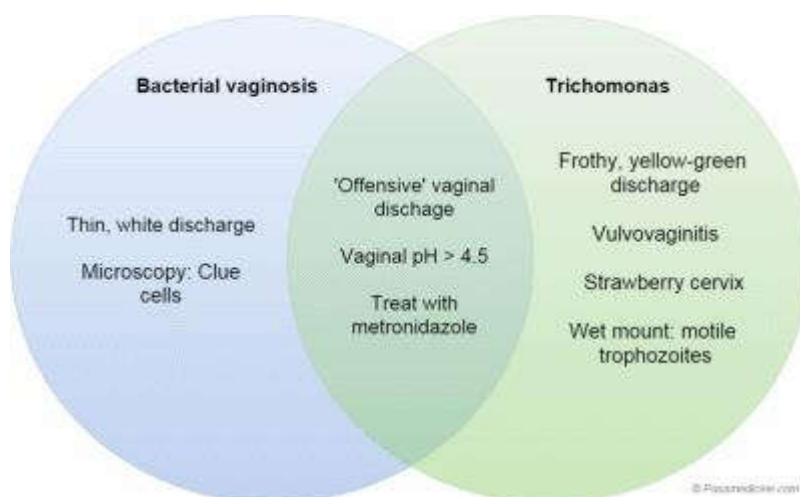
- vaginal discharge: 'fishy', offensive
- asymptomatic in 50%

Amsel's criteria for diagnosis of BV - 3 of the following 4 points should be present

- thin, white homogenous discharge
- clue cells on microscopy: stippled vaginal epithelial cells
- vaginal pH > 4.5
- positive whiff test (addition of potassium hydroxide results in fishy odour)

Management

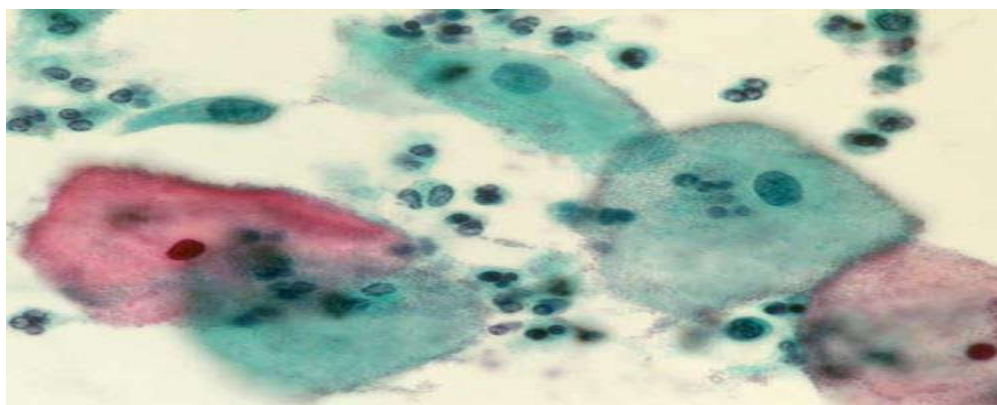
- oral metronidazole for 5-7 days
- 70-80% initial cure rate
- relapse rate > 50% within 3 months
- the BNF suggests topical metronidazole or topical clindamycin as alternatives



Comparison of bacterial vaginosis and *Trichomonas vaginalis*

Bacterial vaginosis in pregnancy

- results in an increased risk of preterm labour, low birth weight and chorioamnionitis, late miscarriage
- it was previously taught that oral metronidazole should be avoided in the first trimester and topical clindamycin used instead. Recent guidelines however recommend that oral metronidazole is used throughout pregnancy. The BNF still advises against the use of high dose metronidazole regimes



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Clue cells - epithelial cells develop a stippled appearance due to being covered with bacteria

External Links

[Clinical Knowledge Summaries](#)

Management of bacterial vaginosis

Brucellosis

Brucellosis is a zoonosis more common in the Middle East and in farmers. Four major species cause infection in humans: *B melitensis* (sheep), *B abortus* (cattle), *B canis* and *B suis* (pigs).

Brucellosis has an incubation period 2 - 6 weeks

Features

- non-specific: fever, malaise
- hepatosplenomegaly
- sacroilitis: spinal tenderness may be seen
- complications: osteomyelitis, infective endocarditis, meningoencephalitis, orchitis
- leukopenia often seen

Diagnosis

- the Rose Bengal plate test can be used for screening but other tests are required to confirm the diagnosis
- Brucella serology is the best test for diagnosis
- blood and bone marrow cultures may be suitable in certain patients, but these tests are often negative

Management

- doxycycline and streptomycin

External Links

[WHO](#)

Brucellosis guidelines

Campylobacter

Campylobacter is the commonest bacterial cause of infectious intestinal disease in the UK. The majority of cases are caused by the Gram-negative bacillus *Campylobacter jejuni*. It is spread by the faecal-oral route and has an incubation period of 1-6 days.

Features:

- prodrome: headache malaise
- diarrhoea: often bloody
- abdominal pain

Management:

- usually self-limiting
- the BNF advises treatment if severe or the patient is immunocompromised. Clinical Knowledge summaries also recommend antibiotics if severe symptoms (high fever, bloody diarrhoea, or more than eight stools per day) or symptoms have last more than one week
- the first-line antibiotic is clarithromycin

Complications:

- Guillain-Barre syndrome may follow *Campylobacter jejuni* infections
- Reiter's syndrome
- septicaemia, endocarditis, arthritis

External Links

[Clinical Knowledge Summaries](#)

Gastroenteritis

Cat scratch disease

Cat scratch disease is generally caused by the Gram negative rod *Bartonella henselae*

Features:

- fever
- history of a cat scratch
- regional lymphadenopathy
- headache, malaise

External Links

[Centers for Disease Control and Prevention](#)

Bartonella

Cellulitis

Cellulitis is a term used to describe an inflammation of the skin and subcutaneous tissues, typically due to infection by *Streptococcus pyogenes* or *Staphylococcus aureus*.

Features

- commonly occurs on the shins
- erythema, pain, swelling
- there may be some associated systemic upset such as fever

Criteria for admission:

NICE Clinical Knowledge Summaries recommend we use the Eron classification to guide how we manage patients with cellulitis:

Class	Features
I	There are no signs of systemic toxicity and the person has no uncontrolled co-morbidities
II	The person is either systemically unwell or systemically well but with a co-morbidity (for example peripheral arterial disease, chronic venous insufficiency, or morbid obesity) which may complicate or delay resolution of infection
III	The person has significant systemic upset such as acute confusion, tachycardia, tachypnoea, hypotension, or unstable co-morbidities that may interfere with a response to treatment, or a limb-threatening infection due to vascular compromise
IV	The person has sepsis syndrome or a severe life-threatening infection such as necrotizing fasciitis

They recommend the following that we admit for intravenous antibiotics the following patients:

- Has Eron Class III or Class IV cellulitis.
- Has severe or rapidly deteriorating cellulitis (for example extensive areas of skin).
- Is very young (under 1 year of age) or frail.
- Is immunocompromized.
- Has significant lymphoedema.
- Has facial cellulitis (unless very mild) or periorbital cellulitis.

The following is recommend regarding Eron Class II cellulitis:

Admission may not be necessary if the facilities and expertise are available in the community to give intravenous antibiotics and monitor the person - check local guidelines.

Other patients can be treated with oral antibiotics.

Management:

- ◆The BNF recommends flucloxacillin as first-line treatment for mild/moderate cellulitis. Clarithromycin or clindamycin is recommended in patients allergic to penicillin.
- ◆Many local protocols now suggest the use of oral clindamycin in patients who have failed to respond to flucloxacillin.

Severe cellulitis should be treated with intravenous benzylpenicillin + flucloxacillin.



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External Links

[Clinical Knowledge Summaries](#)

Cellulitis guidelines

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is reactivation of dormant virus in dorsal root ganglion

Chickenpox is highly infectious:

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults):

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive:

- keep cool, trim nails
- calamine lotion
- school exclusion: the 2016 Public Health England guidelines reverted to the traditional advice to exclude children **until all vesicles have crusted over**, rather than 5 days post onset of rash, which had been advocated over recent years
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered.

A common complication is secondary bacterial infection of the lesions. Rare complications include:

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen.



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Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

*it was traditionally taught that patients were infective until all lesions had scabbed over

External Links

[Department of Health](#)

Greenbook guidelines

[Public Health England](#)

2016 Guidance on Infection Control in Schools

Chickenpox exposure in pregnancy

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is reactivation of dormant virus in dorsal root ganglion. In pregnancy there is a risk to both the mother and also the fetus, a syndrome now termed fetal varicella syndrome

Risks to the mother

- 5 times greater risk of pneumonitis

Fetal varicella syndrome (FVS)

- risk of FVS following maternal varicella exposure is around 1% if occurs before 20 weeks gestation
- studies have shown a very small number of cases occurring between 20-28 weeks gestation and none following 28 weeks
- features of FVS include skin scarring, eye defects (microphthalmia), limb hypoplasia, microcephaly and learning disabilities

Other risks to the fetus

- shingles in infancy: 1-2% risk if maternal exposure in the second or third trimester
- severe neonatal varicella: if mother develops rash between 5 days before and 2 days after birth there is a risk of neonatal varicella, which may be fatal to the newborn child in around 20% of cases

Management of chickenpox exposure

- if there is any doubt about the mother previously having chickenpox maternal blood should be urgently checked for varicella antibodies
- if the pregnant woman is not immune to varicella she should be given varicella zoster immunoglobulin (VZIG) as soon as possible. RCOG and Greenbook guidelines suggest VZIG is effective up to 10 days post exposure
- consensus guidelines suggest oral aciclovir should be given if pregnant women with chickenpox present within 24 hours of onset of the rash

External Links

[RCOG](#)

Chickenpox in pregnancy guidelines

[Greenbook](#)

Varicella

Chikungunya

Alphavirus disease caused by infected mosquitoes. Areas affected are Africa, Asia and Indian subcontinent but in recent years there has been seen in a few cases in Southern Europe. Tanzania had the first reported case.

Symptoms: Prominent symptoms are severe joint pain and abrupt onset of high fever. Other symptoms include general flu-like illness of muscle ache, headache, and fatigue. The disease shares its symptoms with dengue but tends to have more joint pain which can be debilitating. A rash may develop as with other viral illness and swelling of the joints is not uncommon.

Treatment: Relief of symptoms. No specific treatment.

Chlamydia

Chlamydia is the most prevalent sexually transmitted infection in the UK and is caused by *Chlamydia trachomatis*, an obligate intracellular pathogen. Approximately 1 in 10 young women in the UK have *Chlamydia*. The incubation period is around 7-21 days, although it should be remembered a large percentage of cases are asymptomatic

Features

- asymptomatic in around 70% of women and 50% of men
- women: cervicitis (discharge, bleeding), dysuria
- men: urethral discharge, dysuria

Potential complications:

- epididymitis
- pelvic inflammatory disease
- endometritis
- increased incidence of ectopic pregnancies
- infertility
- reactive arthritis
- perihepatitis (Fitz-Hugh-Curtis syndrome)

Investigation:

- traditional cell culture is no longer widely used
- nuclear acid amplification tests (NAATs) are now rapidly emerging as the investigation of choice
- urine (first void urine sample), vulvovaginal swab or cervical swab may be tested using the NAAT technique

Screening

- in England the National *Chlamydia* Screening Programme is open to all men and women aged 15-24 years
- the 2009 SIGN guidelines support this approach, suggesting screening all sexually active patients aged 15-24 years
- relies heavily on opportunistic testing



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Pap smear demonstrating infected endocervical cells. Red inclusion bodies are typical

Management:

- doxycycline (7 day course) or azithromycin (single dose). The 2009 SIGN guidelines suggest azithromycin should be used first-line due to potentially poor compliance with a 7 day course of doxycycline
- if pregnant then azithromycin, erythromycin or amoxicillin may be used. The SIGN guidelines suggest azithromycin 1g stat is the drug of choice 'following discussion of the balance of benefits and risks with the patient'
- patients diagnosed with *Chlamydia* should be offered a choice of provider for initial partner notification - either trained practice nurses with support from GUM, or referral to GUM.
- for men with urethral symptoms: all contacts since, and in the four weeks prior to, the onset of symptoms.
- for women and asymptomatic men all partners from the last six months or the most recent sexual partner should be contacted
- contacts of confirmed *Chlamydia* cases should be offered treatment prior to the results of their investigations being known (treat then test)



Image sourced from [Wikipedia](#)© Image used on license from PathoPic 🔍

Another Pap smear demonstrating infected endocervical cells. Stained with H&E

External Links

[Clinical Knowledge Summaries](#)

Chlamydia guidelines

[SIGN](#)

Management of Chlamydia

[British Association for Sexual Health and HIV](#)

2015 UK national guideline for the management of infection with Chlamydia trachomatis

[Public Health England](#)

Sexually transmitted infections and chlamydia screening in England, 2014

Cholera

Overview

- caused by Vibrio cholerae - Gram negative bacteria

Features

- profuse 'rice water' diarrhoea
- dehydration
- hypoglycaemia

Management

- oral rehydration therapy
 - antibiotics: doxycycline, ciprofloxacin
-

Classification of bacteria

Remember:

- Gram positive cocci = staphylococci + streptococci (including enterococci)
- Gram negative cocci = *Neisseria meningitidis* + *Neisseria gonorrhoeae*, also *Moraxella*

Therefore, **only a small list of Gram positive rods (bacilli) need to be memorised to categorise all bacteria - mnemonic = ABCD L**

- *Actinomyces*
 - *Bacillus anthracis* (anthrax)
 - *Clostridium*
 - Diphtheria: *Corynebacterium diphtheriae*
 - *Listeria monocytogenes*
- Remaining organisms are Gram negative rods

Congenital infections:

♦The major congenital infections encountered in examinations are rubella, toxoplasmosis and cytomegalovirus

♦Cytomegalovirus is the most common congenital infection in the UK. Maternal infection is usually asymptomatic.

	Rubella	Toxoplasmosis	Cytomegalovirus
Characteristic features	Sensorineural deafness Congenital cataracts Congenital heart disease (e.g. patent ductus arteriosus) Glaucoma	Cerebral calcification Chorioretinitis Hydrocephalus	Growth retardation Purpuric skin lesions
Other features	Growth retardation Hepatosplenomegaly Purpuric skin lesions 'Salt and pepper' chorioretinitis Microphthalmia Cerebral palsy	Anaemia Hepatosplenomegaly Cerebral palsy	Sensorineural deafness Encephalitis/seizures Pneumonitis Hepatosplenomegaly Anaemia Jaundice Cerebral palsy

Dengue fever

Dengue fever is a viral infection which can progress to viral haemorrhagic fever (also yellow fever, Lassa fever, Ebola)

Basics

- transmitted by the Aedes aegypti mosquito
- incubation period of 7 days
- a form of disseminated intravascular coagulation (DIC) known as dengue haemorrhagic fever (DHF) may develop. Around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Features

- causes headache (often retro-orbital)
- fever
- myalgia
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash

Treatment is entirely symptomatic e.g. fluid resuscitation, blood transfusion etc

External Links

[Royal College of Physicians](#)

2011 Dengue fever review

[Postgraduate Medical Journal](#)

Review of dengue fever

DNA viruses

Family	Structure	Envelope	Examples/importance
Herpesviruses	Double-stranded, linear	Enveloped	Herpes simplex virus, varicella-zoster virus, cytomegalovirus, Epstein-Barr virus
Hepadnavirus	Double-stranded, circular	Enveloped	Hepatitis B virus
Adenovirus	Double-stranded, linear	Naked	Common cold, conjunctivitis
Parvovirus	Single-stranded , linear	Naked	Parvovirus B19
Papillomavirus	Double-stranded, circular	Naked	HPV
Poxvirus	Double-stranded, linear	Enveloped	Smallpox, molluscum contagiosum
Polyomavirus	Double-stranded, circular	Naked	JC virus (progressive multifocal leukoencephalopathy)

Parvovirus is the only DNA virus that is not double-stranded

HHAPPPPy! - Hepadna; Herpes; Adeno; Pox; Parvo; Papilloma; Polyoma

Epididymo-orchitis

Epididymo-orchitis describes an infection of the epididymis +/- testes resulting in pain and swelling. It is most commonly caused by local spread of infections from the genital tract (such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae*) or the bladder.

The most important differential diagnosis is testicular torsion. This needs to be excluded urgently to prevent ischaemia of the testicle.

Features

- unilateral testicular pain and swelling
- urethral discharge may be present, but urethritis is often asymptomatic
- factors suggesting testicular torsion include patients < 20 years, severe pain and an acute onset

Management

- the British Association for Sexual Health and HIV (BASHH) produced guidelines in 2010
- if the organism is unknown BASHH recommend: ceftriaxone 500mg intramuscularly single dose, plus doxycycline 100mg by mouth twice daily for 10-14 days
- further investigations following treatment are recommended to exclude any underlying structural abnormalities

External Links

[British Association for Sexual Health and HIV](#)

2010 Epididymo-orchitis guidelines

Epstein-Barr virus: associated conditions

Malignancies associated with EBV infection:

- Burkitt's lymphoma*
- Hodgkin's lymphoma
- nasopharyngeal carcinoma
- HIV-associated central nervous system lymphomas

The non-malignant condition hairy leukoplakia is also associated with EBV infection.

*EBV is currently thought to be associated with both African and sporadic Burkitt's

Escherichia coli

Escherichia coli is a facultative anaerobic, lactose-fermenting, Gram negative rod which is a normal gut commensal.

***E. coli* infections lead to a variety of diseases in humans including:**

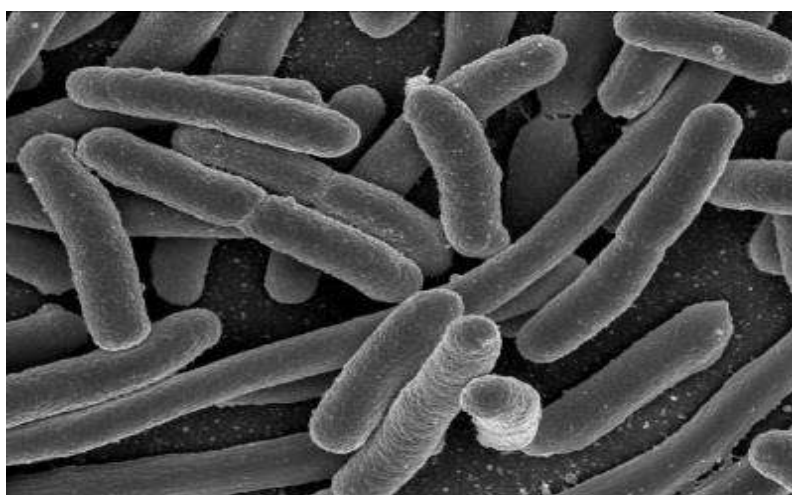
- diarrhoeal illnesses
- UTIs
- neonatal meningitis

Serotypes

***E. coli* may be classified according to the antigens which may trigger an immune response:**

Antigen	Origin	Notes
O	Lipopolysaccharide layer	
K	Capsule	Neonatal meningitis secondary to <i>E. coli</i> is usually caused by a serotype that contains the capsular antigen K-1
H	Flagellin	

E. coli O157:H7 is a particular strain associated with severe, haemorrhagic, watery diarrhoea. It has a high mortality rate and can be complicated by haemolytic uraemic syndrome. It is often spread by contaminated ground beef.



Scanning electron micrograph of *Escherichia coli*, grown in culture and adhered to a cover slip.
Credit: NIAID

Gastroenteritis

♦Gastroenteritis may either occur whilst at home or whilst travelling abroad (travellers' diarrhoea)

♦**Travellers' diarrhoea** may be defined as at least 3 loose to watery stools in 24 hours with or without one or more of abdominal cramps, fever, nausea, vomiting or blood in the stool. The most common cause is *Escherichia coli*

♦Another pattern of illness is 'acute food poisoning'. This describes the sudden onset of nausea, vomiting and diarrhoea after the ingestion of a toxin. Acute food poisoning is typically caused by *Staphylococcus aureus*, *Bacillus cereus* or *Clostridium perfringens*.

Stereotypical histories

Infection	Typical presentation
<i>Escherichia coli</i>	Common amongst travellers Watery stools Abdominal cramps and nausea
Giardiasis	Prolonged, non-bloody diarrhoea
Cholera	Profuse, watery diarrhoea Severe dehydration resulting in weight loss Not common amongst travellers
<i>Shigella</i>	Bloody diarrhoea Vomiting and abdominal pain
<i>Staphylococcus aureus</i>	Severe vomiting Short incubation period
<i>Campylobacter</i>	A flu-like prodrome is usually followed by crampy abdominal pains, fever and diarrhoea which may be bloody Complications include Guillain-Barre syndrome
<i>Bacillus cereus</i>	Two types of illness are seen • vomiting within 6 hours, stereotypically due to rice • diarrhoeal illness occurring after 6 hours
Amoebiasis	Gradual onset bloody diarrhoea, abdominal pain and tenderness which may last for several weeks

Incubation period

- 1-6 hrs: *Staphylococcus aureus*, *Bacillus cereus**
- 12-48 hrs: *Salmonella*, *Escherichia coli*
- 48-72 hrs: *Shigella*, *Campylobacter*
- > 7 days: Giardiasis, Amoebiasis

*vomiting subtype, the diarrhoeal illness has an incubation period of 6-14 hours

External Links

[Royal College of Physicians](#)

2011 Diarrhoeal disease review

Genital warts

Genital warts (also known as condylomata accuminata) are a common cause of attendance at genitourinary clinics. They are caused by the many varieties of the human papilloma virus HPV, especially types 6 & 11. It is now well established that HPV (primarily types 16,18 & 33) predisposes to cervical cancer.

Features

- small (2 - 5 mm) fleshy protuberances which are slightly pigmented
- may bleed or itch

Management

- topical podophyllum or cryotherapy are commonly used as first-line treatments depending on the location and type of lesion. Multiple, non-keratinised warts are generally best treated with topical agents whereas solitary, keratinised warts respond better to cryotherapy
- imiquimod is a topical cream which is generally used second line
- genital warts are often resistant to treatment and recurrence is common although the majority of anogenital infections with HPV clear without intervention within 1-2 years

External Links

[British Association for Sexual Health and HIV](#)

United Kingdom National Guideline on the Management of Anogenital Warts

[Clinical Knowledge Summaries](#)

Ano-genital warts guidelines

Giardiasis

Giardiasis is caused by the flagellate protozoan *Giardia lamblia*. It is spread by the faeco-oral route

Features

- often asymptomatic
- lethargy, bloating, abdominal pain
- non-bloody diarrhoea
- chronic diarrhoea, malabsorption and lactose intolerance can occur
- stool microscopy for trophozoite and cysts are classically negative, therefore duodenal fluid aspirates or 'string tests' (fluid absorbed onto swallowed string) are sometimes needed

Treatment is with metronidazole

Gonorrhoea

Gonorrhoea is caused by the Gram negative diplococcus *Neisseria gonorrhoeae*. Acute infection can occur on any mucous membrane surface, typically genitourinary but also rectum and pharynx. The incubation period of gonorrhoea is 2-5 days

Features

- males: urethral discharge, dysuria
- females: cervicitis e.g. leading to vaginal discharge
- rectal and pharyngeal infection is usually asymptomatic.

Microbiology

- immunisation is not possible and reinfection is common due to antigen variation of type IV pili (proteins which adhere to surfaces) and Opa proteins (surface proteins which bind to receptors on immune cells)

Local complications that may develop include urethral strictures, epididymitis and salpingitis (hence may lead to infertility). Disseminated infection may occur - see below.

Management

- **ciprofloxacin** used to be the treatment of choice. However, there is increased resistance to ciprofloxacin and therefore cephalosporins are now used
- the 2011 British Society for Sexual Health and HIV (BASHH) guidelines recommend ceftriaxone 500 mg intramuscularly as a single dose with azithromycin 1 g oral as a single dose. The azithromycin is thought to act synergistically with ceftriaxone and is also useful for eradicating any co-existent Chlamydia infections. This combination can be used in pregnant women as well
- if **ceftriaxone** is refused or contraindicated other options include cefixime 400mg PO (single dose)



Colorized scanning electron micrograph of *Neisseria gonorrhoeae*. Credit: NIAID.

Disseminated gonococcal infection (DGI) and gonococcal arthritis may also occur, with gonococcal infection being the most common cause of septic arthritis in young adults. The pathophysiology of DGI is not fully understood but is thought to be due to haematogenous spread from mucosal infection (e.g. Asymptomatic genital infection). Initially there may be a classic triad of symptoms: tenosynovitis, migratory polyarthritis and dermatitis. Later complications include septic arthritis, endocarditis and perihepatitis (Fitz-Hugh-Curtis syndrome).

Key features of disseminated gonococcal infection

- tenosynovitis
- migratory polyarthrititis
- dermatitis (lesions can be maculopapular or vesicular)

External Links

[British Society for Sexual Health and HIV \(BASHH\)](#)

2011 Gonorrhoea guidelines

[Clinical Knowledge Summaries](#)

Gonorrhea guidelines

H1N1 influenza pandemic

The 2009 H1N1 influenza (swine flu) outbreak was first observed in Mexico in early 2009. In June 2009, the WHO declared the outbreak to be a pandemic.

H1N1

The H1N1 virus is a subtype of the influenza A virus and the most common cause of flu in humans. The 2009 pandemic was caused by a new strain of the H1N1 virus.

The following groups are particularly at risk:

- patients with chronic illnesses and those on immunosuppressants
- pregnant women
- young children under 5 years old.

Features:

The majority of symptoms are typical of those seen in a flu-like illness:

- fever greater than 38°C
- myalgia
- lethargy
- headache
- rhinitis
- sore throat
- cough
- diarrhoea and vomiting

♦A minority of patients may go on to develop an acute respiratory distress syndrome which may require ventilatory support.

Treatment

There are two main treatments currently available:

Oseltamivir (Tamiflu)

- oral medication
- a neuraminidase inhibitor which prevents new viral particles from being released by infected cells
- common side-effects include nausea, vomiting, diarrhoea and headaches

Zanamivir (Relenza)

- inhaled medication*
- also a neuraminidase inhibitor
- may induce bronchospasm in asthmatics

*intravenous preparations are available for patients who are acutely unwell

Hand, foot and mouth disease

Hand, foot and mouth disease is a self-limiting condition affecting children. It is caused by the intestinal viruses of the Picornaviridae family (most commonly coxsackie A16 and enterovirus 71). It is very contagious and typically occurs in outbreaks at nursery

Clinical features

- mild systemic upset: sore throat, fever
- oral ulcers
- followed later by vesicles on the palms and soles of the feet



© Image used on license from [DermNet NZ](#)



© Image used on license from [DermNet NZ](#)



Management

- general advice about hydration and analgesia
- reassurance no link to disease in cattle
- children do not need to be excluded from school*

*The HPA recommends that children who are unwell should be kept off school until they feel better. They also advise that you contact them if you suspect that there may be a large outbreak.

Hepatitis B

Hepatitis B is a double-stranded DNA hepadnavirus and is spread through exposure to infected blood or body fluids, including vertical transmission from mother to child. The incubation period is 6-20 weeks.

The features of hepatitis B include fever, jaundice and elevated liver transaminases.

Complications of hepatitis B infection:

- chronic hepatitis (5-10%)
- fulminant liver failure (1%)
- hepatocellular carcinoma
- glomerulonephritis
- polyarteritis nodosa
- cryoglobulinaemia

Immunisation against hepatitis B (please see the Greenbook link for more details):

- contains HBsAg adsorbed onto aluminium hydroxide adjuvant and is prepared from yeast cells using recombinant DNA technology
- most schedules give 3 doses of the vaccine with a recommendation for a one-off booster 5 years following the initial primary vaccination
- at risk groups who should be vaccinated include: healthcare workers, intravenous drug users, sex workers, close family contacts of an individual with hepatitis B, individuals receiving blood transfusions regularly, chronic kidney disease patients who may soon require renal replacement therapy, prisoners, chronic liver disease patients
- around 10-15% of adults fail to respond or respond poorly to 3 doses of the vaccine. Risk factors include age over 40 years, obesity, smoking, alcohol excess and immunosuppression
- testing for anti-HBs is only recommended for those at risk of occupational exposure (i.e. Healthcare workers) and patients with chronic kidney disease. In these patients anti-HBs levels should be checked 1-4 months after primary immunization.

- the table below shows how to interpret anti-HBs levels:

Anti-HBs level (mIU/ml)	Response
> 100	Indicates adequate response, no further testing required. Should still receive booster at 5 years
10 - 100	Suboptimal response - one additional vaccine dose should be given. If immunocompetent no further testing is required
< 10	Non-responder. Test for current or past infection. Give further vaccine course (i.e. 3 doses again) with testing following. If still fails to respond then HBIG would be required for protection if exposed to the virus

Management of hepatitis B

- pegylated interferon-alpha used to be the only treatment available. It reduces viral replication in up to 30% of chronic carriers. A better response is predicted by being female, < 50 years old, low HBV DNA levels, non-Asian, HIV negative, high degree of inflammation on liver biopsy
- whilst NICE still advocate the use of pegylated interferon first-line other antiviral medications are increasingly used with an aim to suppress viral replication (not in a dissimilar way to treating HIV patients)
- examples include tenofovir and entecavir

External Links

[Greenbook](#)

Hepatitis B

[NICE](#)

2013 Chronic hepatitis B guidelines

Hepatitis B and pregnancy

Basics

- all pregnant women are offered screening for hepatitis B
- babies born to mothers who are chronically infected with hepatitis B or to mothers who've had acute hepatitis B during pregnancy should receive a complete course of vaccination + hepatitis B immunoglobulin
- studies are currently evaluating the role of oral antiviral treatment (e.g. Lamivudine) in the latter part of pregnancy
- there is little evidence to suggest caesarean section reduces vertical transmission rates
- hepatitis B cannot be transmitted via breastfeeding (in contrast to HIV)

External Links

[Green Book](#)

Hepatitis B guidelines

[Hepatitis B Foundation](#)

Hepatitis B in pregnancy

Hepatitis B serology

Interpreting hepatitis B serology is a dying art form which still occurs at regular intervals in medical exams. **It is important to remember a few key facts:**

- surface antigen (HBsAg) is the first marker to appear and causes the production of anti-HBs
- HBsAg normally implies acute disease (present for 1-6 months)
- if HBsAg is present for > 6 months then this implies chronic disease (i.e. Infective)
- Anti-HBs implies immunity (either exposure or immunisation). It is negative in chronic disease
- Anti-HBc implies previous (or current) infection. IgM anti-HBc appears during acute or recent hepatitis B infection and is present for about 6 months. IgG anti-HBc persists
- HbeAg results from breakdown of core antigen from infected liver cells as is therefore a marker of infectivity.

Example results

- previous immunisation: anti-HBs positive, all others negative
- previous hepatitis B (> 6 months ago), not a carrier: anti-HBc positive, HBsAg negative
- previous hepatitis B, now a carrier: anti-HBc positive, HBsAg positive

HBsAg = ongoing infection, either acute or chronic if present > 6 months

anti-HBc = caught, i.e. negative if immunized

External Links

[Centers for Disease Control and Prevention](#)

Interpretation of Hepatitis B Serologic Test Results

Hepatitis E

Overview

- RNA hepevirus
- spread by the faecal-oral route
- incubation period: 3-8 weeks
- common in Central and South-East Asia, North and West Africa, and in Mexico
- causes a similar disease to hepatitis A, but carries a significant mortality (about 20%) during pregnancy
- does not cause chronic disease or an increased risk of hepatocellular cancer
- a vaccine is currently in development*, but is not yet in widespread use

*New England Journal of Medicine 356:895, 2007

External Links

[Centers for Disease Control and Prevention](#)

Hepatitis E review

HIV and pregnancy

With the increased incidence of HIV infection amongst the heterosexual population there are an increasing number of HIV positive women giving birth in the UK. In London the incidence may be as high as 0.4% of pregnant women. The aim of treating HIV positive women during pregnancy is to minimise harm to both the mother and fetus, and to reduce the chance of vertical transmission.

Guidelines regularly change on this subject and most recent guidelines can be found using the links provided.

Factors which reduce vertical transmission (from 25-30% to 2%):

- maternal antiretroviral therapy
- mode of delivery (caesarean section)
- neonatal antiretroviral therapy
- infant feeding (bottle feeding)

Screening

- NICE guidelines recommend offering HIV screening to all pregnant women

Antiretroviral therapy

- all pregnant women should be offered antiretroviral therapy regardless of whether they were taking it previously

Mode of delivery

- vaginal delivery is recommended if viral load is less than 50 copies/ml at 36 weeks, otherwise caesarian section is recommended
- a zidovudine infusion should be started four hours before beginning the caesarean section

Neonatal antiretroviral therapy

- zidovudine is usually administered orally to the neonate if maternal viral load is <50 copies/ml. Otherwise triple ART should be used. Therapy should be continued for 4-6 weeks.

Infant feeding

- in the UK all women should be advised not to breast feed

External Links

[BHIVA](#)

2014 HIV in pregnancy guidelines

[BHIVA](#)

2013 Useful patient orientated leaflet

[Patient.info](#)

HIV and pregnancy

HIV: diarrhea

Diarrhoea is common in patients with HIV. This may be due to the effects of the virus itself (HIV enteritis) or opportunistic infections

Possible causes:

- Cryptosporidium + other protozoa (most common)
- Cytomegalovirus
- *Mycobacterium avium intracellulare*
- Giardia

Cryptosporidium is the most common infective cause of diarrhoea in HIV patients. It is an intracellular protozoa and has an incubation period of 7 days. Presentation is very variable, ranging from mild to severe diarrhoea. A modified Ziehl-Neelsen stain (acid-fast stain) of the stool may reveal the characteristic red cysts of Cryptosporidium. Treatment is difficult, with the mainstay of management being supportive therapy*

Mycobacterium avium intracellulare is an atypical mycobacteria seen with the CD4 count is below 50. Typical features include fever, sweats, abdominal pain and diarrhoea. There may be hepatomegaly and deranged LFTs. Diagnosis is made by blood cultures and bone marrow examination. Management is with rifabutin, ethambutol and clarithromycin

*nitazoxanide is licensed in the US for immunocompetent patients

HIV: immunisation

The Department of Health 'Greenbook' on immunisation defers to the British HIV Association for guidelines relating to immunisation of HIV-infected adults

Vaccines that can be used in all HIV-infected adults	Vaccines that can be used if CD4 > 200	Contraindicated in HIV-infected adults
Hepatitis A Hepatitis B <i>Haemophilus influenzae</i> B (Hib) Influenza-parenteral Japanese encephalitis Meningococcus-MenC Meningococcus-ACWY I Pneumococcus-PPV23 Poliomyelitis-parenteral (IPV) Rabies Tetanus-Diphtheria (Td)	Measles, Mumps, Rubella (MMR) Varicella Yellow Fever	Cholera CVD103-HgR Influenza-intranasal Poliomyelitis-oral (OPV) Tuberculosis (BCG)

External Links

[BHIVA](#)

2008 HIV immunisation guidelines

HIV: Kaposi's sarcoma

Kaposi's sarcoma

- caused by HHV-8 (human herpes virus 8)
- presents as purple papules or plaques on the skin or mucosa (e.g. gastrointestinal and respiratory tract)
- skin lesions may later ulcerate
- respiratory involvement may cause massive haemoptysis and pleural effusion
- radiotherapy + resection



Kaposi's sarcoma in a patient with HIV

External Links

DermIS.net

Picture of Kaposi's sarcoma

HIV: Mycobacterium avium complex

Mycobacterium avium complex (MAC) is an atypical mycobacterial infection seen in HIV patients. It is caused by both Mycobacterium avium and Mycobacterium intracellulare, and is often referred to as Mycobacterium avium-intracellulare (MAI). Over 95% of MAC infections in patients with HIV are caused by Mycobacterium avium. MAC is generally seen when the CD4 count is less than 50 cells/mm³

Features

- fever, sweats
- abdominal: pain, diarrhoea
- lung: dyspnoea, cough
- anaemia
- lymphadenopathy
- hepatomegaly/deranged LFTs

Diagnosis

- blood cultures
- bone marrow aspirate

Prophylaxis

- clarithromycin or azithromycin when CD4 is less than 100 cells/mm³

Management

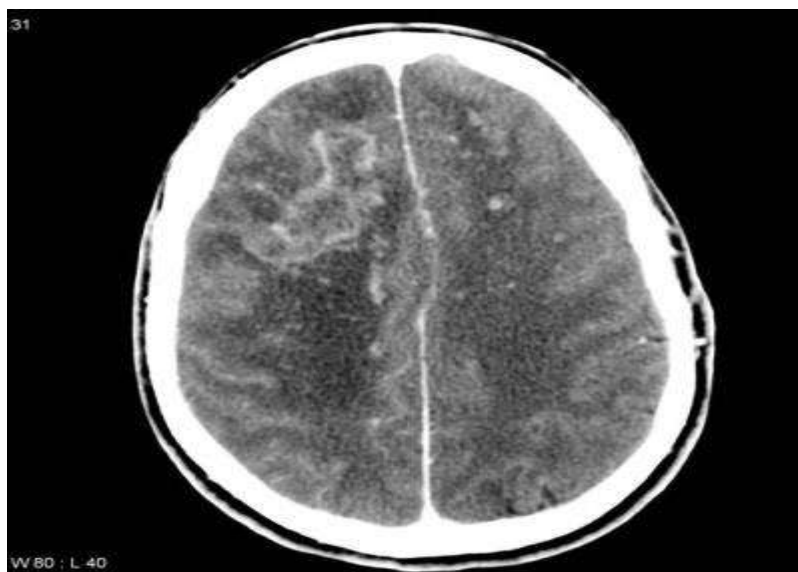
- rifampicin + ethambutol + clarithromycin

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine



© Image used on license from [Radiopaedia](https://radiopaedia.org/)



Cerebral toxoplasmosis: CT scan with contrast showing multiple ring enhancing lesions



© Image used on license from [Radiopaedia](#)



Cerebral toxoplasmosis: MRI (T1 C+) demonstrates multiple small peripherally enhancing nodules located predominantly in the basal ganglia as well as the central portions of the cerebellar hemispheres. Only a small amount of surrounding oedema is present.

Primary CNS lymphoma

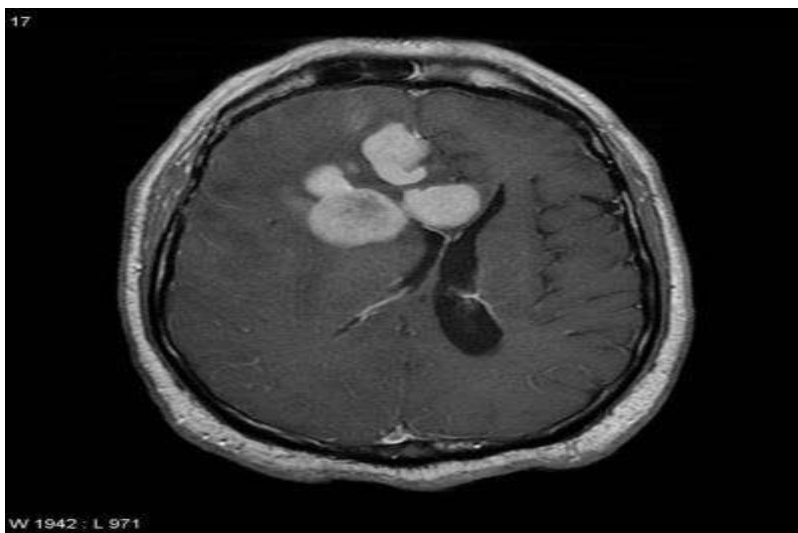
- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours



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Primary CNS lymphoma: Non-contrast CT demonstrates a hyper-attenuating mass adjacent to the left lateral ventricle, with no calcification or haemorrhage.



© Image used on license from [Radiopaedia](#)



Primary CNS lymphoma: MRI (T1 C+) demonstrates a large multilobulated mass in the right frontal lobe. It homogeneously enhances and extends to involve the caudate and the periventricular area. There is significant mass effect.

♦Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	Lymphoma
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF: high opening pressure, India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen.

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy

External Links

[Radiopaedia](#)

Toxoplasmosis vs lymphoma

HIV: opportunistic infections and other disorders

The table below shows the infections and other disorders that may be encountered by patients with HIV according to the CD4 count.

CD4 count 200 - 500 cells/mm³

Disorder	Notes
Oral thrush	Secondary to <i>Candida albicans</i>
Shingles	Secondary to herpes zoster
Hairy leukoplakia	Secondary to EBV
Kaposi sarcoma	Secondary to HHV-8

CD4 count 100 - 200 cells/mm³

Disorder	Notes
Cryptosporidiosis	Whilst patients with a CD4 count of 200-500 may develop cryptosporidiosis the disease is usually self-limiting and similar to that in immunocompetent hosts
Cerebral toxoplasmosis	
Progressive multifocal leukoencephalopathy	Secondary to the JC virus
<i>Pneumocystis jirovecii</i> pneumonia	
HIV dementia	

CD4 count 50 - 100 cells/mm³

Disorder	Notes
Aspergillosis	Secondary to <i>Aspergillus fumigatus</i>
Oesophageal candidiasis	Secondary to <i>Candida albicans</i>
Cryptococcal meningitis	
Primary CNS lymphoma	Secondary to EBV

CD4 count < 50 cells/mm³

Disorder	Notes
Cytomegalovirus retinitis	Affects around 30-40% of patients with CD4 < 50 cells/mm ³
Mycobacterium avium-intracellulare infection	

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm³ should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs.

♦Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause:

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)

Management

- co-trimoxazole
- IV pentamidine in severe cases
- steroids if hypoxic (if $pO_2 < 9.3\text{kPa}$ then steroids reduce risk of respiratory failure by 50% and death by a third).



© Image used on license from [Radiopaedia](#)

CT scan showing a large pneumothorax developing in a patient with *Pneumocystis jiroveci* pneumonia

External Links

[Patient.info](#)

Pneumocystis Jirovecii Pneumonia review

HIV: seroconversion

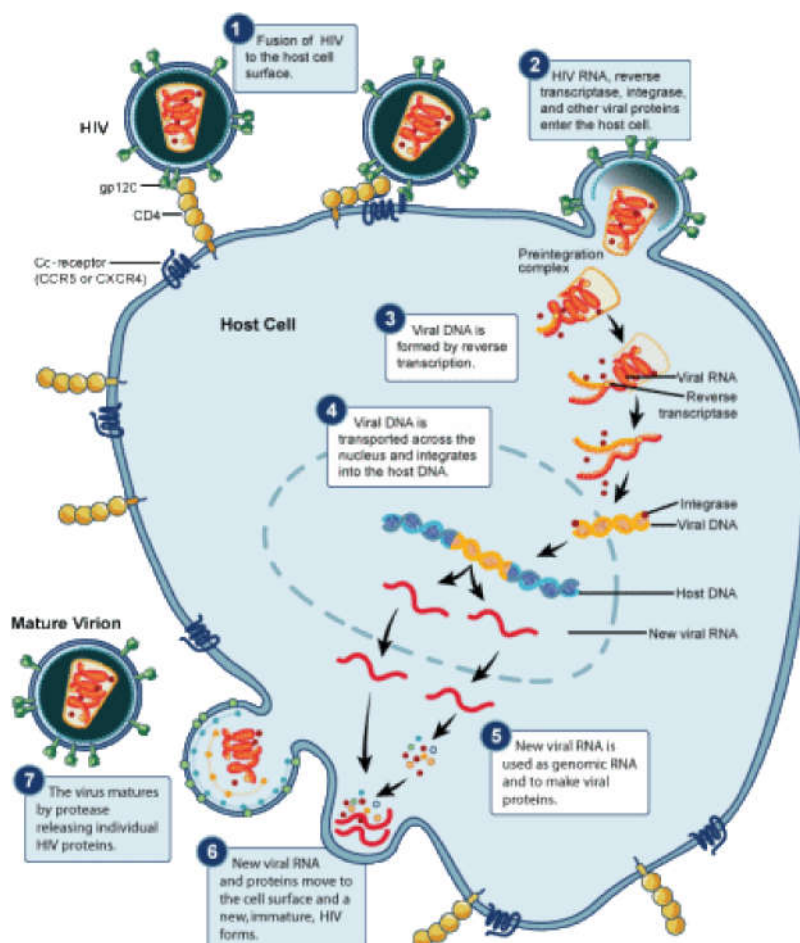
HIV seroconversion is symptomatic in 60-80% of patients and typically presents as a glandular fever type illness. Increased symptomatic severity is associated with poorer long term prognosis. It typically occurs 3-12 weeks after infection

Features

- sore throat
- lymphadenopathy
- malaise, myalgia, arthralgia
- diarrhoea
- maculopapular rash
- mouth ulcers
- rarely meningoencephalitis

Diagnosis

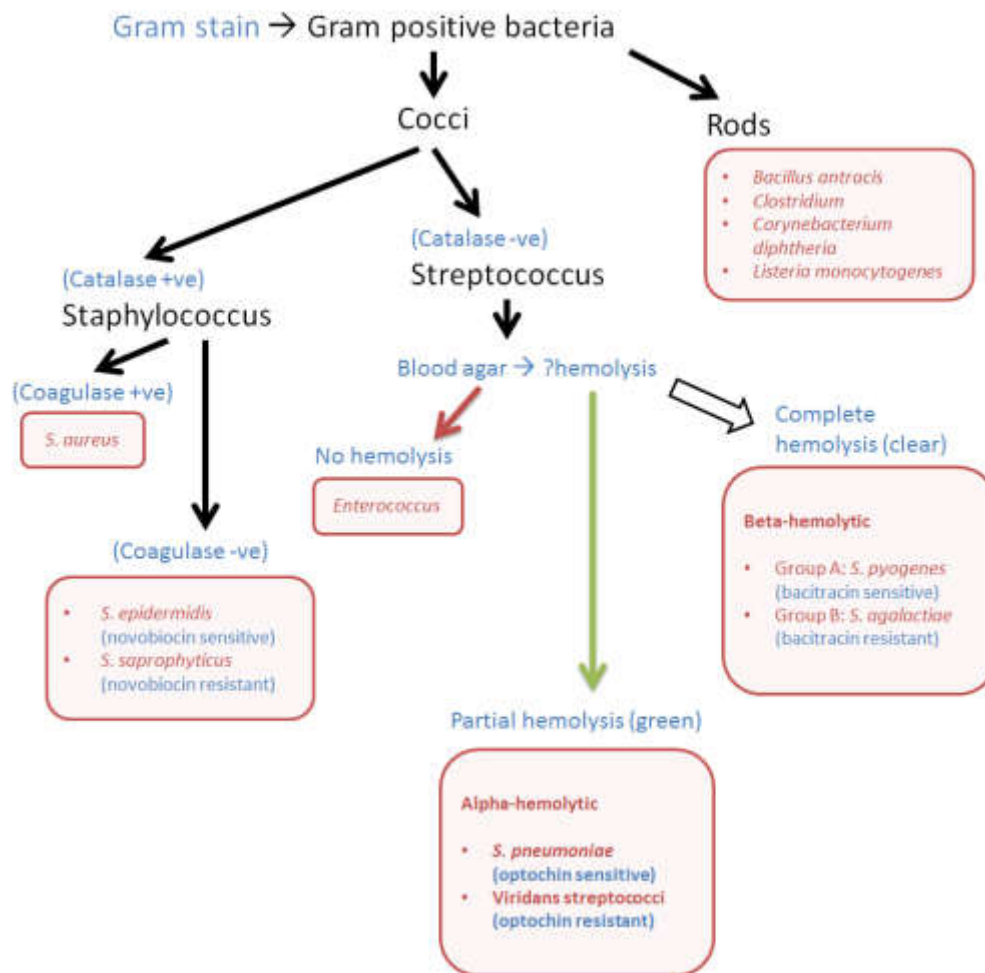
- antibodies to HIV may not be present
- HIV PCR and p24 antigen tests can confirm diagnosis



An illustration model of the HIV Replication Cycle. Each step of the cycle is numbered and concisely described. Credit: NIAID

Identifying gram-positive bacteria

Gram positive bacteria will turn purple/blue following the gram staining. Microscopy will then reveal the shape, either cocci or rods.



Rods (bacilli)

- *Actinomyces*
- *Bacillus anthracis*
- *Clostridium*
- *Corynebacterium diphtheriae*
- *Listeria monocytogenes*

Cocci

- makes catalase: Staphylococci
- does not make catalase: Streptococci

Staphylococci

- makes coagulase: *S. aureus*
- does not make coagulase: *S. epidermidis* (novobiocin sensitive), *S. saprophyticus* (novobiocin resistant)

Streptococci

- partial haemolysis (green colour on blood agar): α -haemolytic
- complete haemolysis (clear): β -haemolytic
- no haemolysis: γ -haemolytic

α -haemolytic streptococci

- optochin sensitive: *S. pneumoniae*
- optochin resistant: Viridans streptococci

β -haemolytic streptococci

- bacitracin sensitive: Group A: *S. pyogenes*
- bacitracin resistant: Group B: *S. agalactiae*

Incubation periods

Questions may either ask directly about incubation periods or they may be used to provide a clue in a differential diagnosis

Less than 1 week

- meningococcus
- diphtheria
- influenza
- scarlet fever

1 - 2 weeks

- malaria
- dengue fever
- typhoid
- measles

2 - 3 weeks

- mumps
- rubella
- chickenpox

Longer than 3 weeks

- infectious mononucleosis
- cytomegalovirus
- viral hepatitis
- HIV

Infectious mononucleosis

Infectious mononucleosis (glandular fever) is caused by the Epstein-Barr virus (also known as human herpesvirus 4, HHV-4). It is most common in adolescents and young adults.

Features

- sore throat
- lymphadenopathy
- pyrexia
- malaise, anorexia, headache
- palatal petechiae
- splenomegaly - occurs in around 50% of patients and may rarely predispose to splenic rupture.
- hepatitis

Chapter: Infectious Diseases & STI

- presence of 50% lymphocytes with at least 10% atypical lymphocytes
- haemolytic anaemia secondary to cold agglutins (IgM)
- a maculopapular, pruritic rash develops in around 99% of patients who take ampicillin/amoxicillin whilst they have infectious mononucleosis

Diagnosis

- heterophil antibody test (Monospot test) - NICE guidelines suggest FBC and Monospot in the second week of the illness to confirm a diagnosis of glandular fever.

Management is supportive and includes:

- rest during the early stages, drink plenty of fluid, avoid alcohol
- simple analgesia for any aches or pains
- consensus guidance in the UK is to avoid playing contact sports for 8 weeks after having glandular fever to reduce the risk of splenic rupture

External Links

[Patient.info](#)

Infectious mononucleosis review

Infective endocarditis: prognosis and management

Poor prognostic factors

- Staph aureus infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism

- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%.

Current antibiotic guidelines (source: British National Formulary)

Scenario	Suggested antibiotic therapy
Initial blind therapy	Native valve - amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis - vancomycin + low-dose gentamicin If prosthetic valve - vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic vancomycin + low-dose gentamicin

Indications for surgery:

- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

External Links

[BNF](#)

Infective endocarditis guidelines

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, usually being spread by sand flies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at site of bite
- may be underlying ulcer

Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopenia secondary to hypersplenism

*occasionally patients may report increased appetite with paradoxical weight loss

External Links

[Royal College of Physicians](#)

2011 Leishmaniasis review

[DermNet NZ](#)

Leishmaniasis review

Leprosy

Leprosy is a granulomatous disease primarily affecting the peripheral nerves and skin. It is caused by *Mycobacterium leprae*.

Features

- patches of hypopigmented skin typically affecting the buttocks, face, and extensor surfaces of limbs
- sensory loss

♦The degree of cell mediated immunity determines the type of leprosy a patient will develop.

♦**Low** degree of cell mediated immunity → **lepromatous** leprosy ('**multibacillary**')

- extensive skin involvement
- symmetrical nerve involvement

♦**High** degree of cell mediated immunity → **tuberculoid** leprosy ('**paucibacillary**')

- limited skin disease
- asymmetric nerve involvement

Management

- WHO-recommended triple therapy: rifampicin, dapsone and clofazimine

External Links

[DermNet NZ](#)

Leprosy

Leptospirosis

Also known as **Weil's disease***, leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in abattoir. It is caused by the spirochaete *Leptospira interrogans* (serogroup L icterohaemorrhagiae), classically being spread by contact with infected rat urine. Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- fever
- flu-like symptoms
- renal failure (seen in 50% of patients)
- jaundice
- subconjunctival haemorrhage
- headache, may herald the onset of meningitis

Management

- high-dose benzylpenicillin or doxycycline

*the term Weil's disease is sometimes reserved for the most severe 10% of cases that are associated with jaundice

Linezolid

Linezolid is a type of oxazolidinone antibiotic which has been introduced in recent years. It inhibits bacterial protein synthesis by stopping formation of the 70s initiation complex and is bacteriostatic nature

Spectrum, highly active against Gram positive organisms including:

- MRSA (Methicillin-resistant *Staphylococcus aureus*)
- VRE (Vancomycin-resistant enterococcus).
- GISA (Glycopeptide Intermediate *Staphylococcus aureus*).

Adverse effects

- thrombocytopenia (reversible on stopping)
- monoamine oxidase inhibitor: avoid tyramine containing foods

Listeria

Listeria monocytogenes is a Gram positive bacillus which has the unusual ability to multiply at low temperatures. It is typically spread via contaminated food, typically unpasteurised dairy products. Infection is particularly dangerous to the unborn child where it can lead to miscarriage.

Features - can present in a variety of ways

- diarrhoea, flu-like illness
- pneumonia , meningoencephalitis
- ataxia and seizures

Suspected Listeria infection should be investigated by taking blood cultures. CSF may reveal a pleocytosis, with 'tumbling motility' on wet mounts

Management

- Listeria is sensitive to amoxicillin/ampicillin (cephalosporins usually inadequate)
- Listeria meningitis should be treated with IV amoxicillin/ampicillin and gentamicin

In pregnant women

- pregnant women are almost 20 times more likely to develop listeriosis compared with the rest of the population due to changes in the immune system
- fetal/neonatal infection can occur both transplacentally and vertically during child birth
- complications include miscarriage, premature labour, stillbirth and chorioamnionitis
- diagnosis can only be made from blood cultures
- treatment is with amoxicillin

External Links

[DermNet NZ](#)

Leprosy

[YouTube](#)

Tumbling motility of listeria

Lyme disease

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks

Features

- early: erythema chronicum migrans + systemic features (fever, arthralgia)
- CVS: heart block, myocarditis
- neuro: cranial nerve palsies, meningitis

Investigation

- serology: antibodies to *Borrelia burgdorferi*

Management

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)

External Links

[Dermnet NZ](#)

Lyme disease

[Clinical Knowledge Summaries](#)

Lyme disease: management

Lyme disease: features

Early features

- erythema chronicum migrans (small papule often at site of the tick bite which develops into a larger annular lesion with central clearing, 'bull's-eye'. Occurs in 70% of patients)
- systemic symptoms: malaise, fever, arthralgia

Later features

- CVS: heart block, myocarditis
 - neurological: cranial nerve palsies, meningitis
 - polyarthritis
-

Lymphadenopathy

There are many causes of generalised lymphadenopathy

Infective

- infectious mononucleosis
- HIV, including seroconversion illness
- eczema with secondary infection
- rubella
- toxoplasmosis
- CMV
- tuberculosis
- roseola infantum

Neoplastic

- leukaemia
- lymphoma

Others:

- autoimmune conditions: SLE, rheumatoid arthritis
 - graft versus host disease
 - sarcoidosis
 - drugs: phenytoin and to a lesser extent allopurinol, isoniazid
-

Malaria

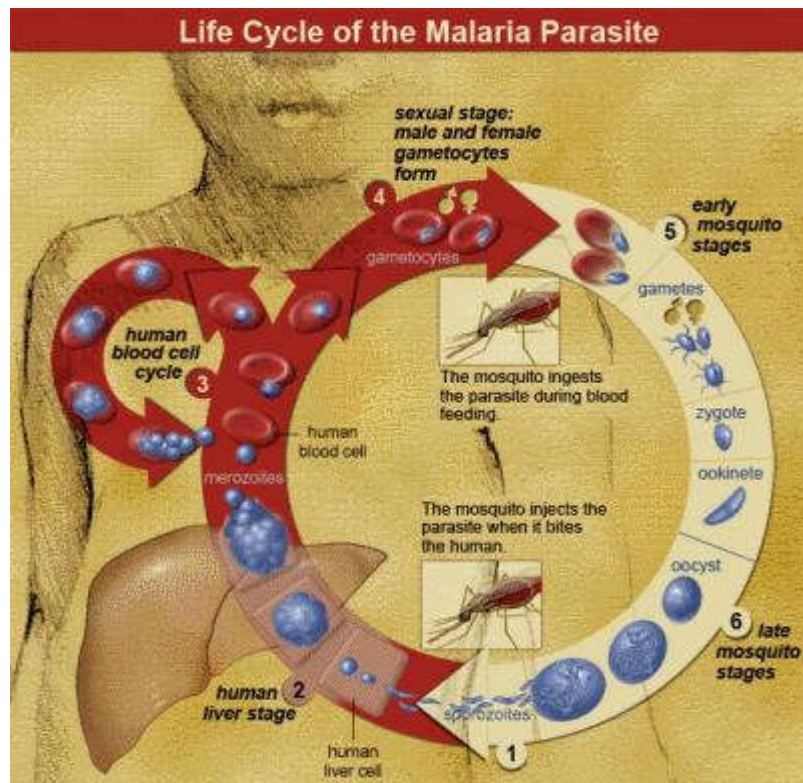
Malaria is a disease caused by *Plasmodium* protozoa which is spread by the female Anopheles mosquito. **There are four different species which cause disease in man:**

- *Plasmodium falciparum*
- *Plasmodium vivax*
- *Plasmodium ovale*
- *Plasmodium malariae*

Plasmodium falciparum causes nearly all episodes of severe malaria. The other three types, of which *Plasmodium vivax* is the most common, cause 'benign' malaria

The protection from malaria that sickle-cell trait offers is well documented. **Other protective factors include:**

- G6PD deficiency
- HLA-B53
- absence of Duffy antigens



An illustration of the life cycle of the malaria parasite. Credit: NIAID

Malaria: Falciparum

Feature of severe malaria

- schizonts on a blood film
- parasitaemia $> 2\%$
- hypoglycaemia
- acidosis
- temperature $> 39^{\circ}\text{C}$
- severe anaemia
- complications as below.

Complications

- cerebral malaria: seizures, coma
- acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown
- acute respiratory distress syndrome (ARDS)
- hypoglycaemia
- disseminated intravascular coagulation (DIC)

Uncomplicated falciparum malaria

- strains resistant to chloroquine are prevalent in certain areas of Asia and Africa
- the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy
- examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperazine

Severe falciparum malaria

- a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state
- intravenous artesunate is now recommended by WHO in preference to intravenous quinine
- if parasite count > 10% then exchange transfusion should be considered
- shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse

External Links

[WHO](#)

Malaria guidelines

Malaria: non-falciparum

The most common cause of non-falciparum malaria is *Plasmodium vivax*, with *Plasmodium ovale* and *Plasmodium malariae* accounting for the other cases. *Plasmodium vivax* is often found in Central America and the Indian Subcontinent whilst *Plasmodium ovale* typically comes from Africa

Features

- general features of malaria: fever, headache, splenomegaly
- *Plasmodium vivax/ovale*: cyclical fever every 48 hours. *Plasmodium malariae*: cyclical fever every 72 hours
- *Plasmodium malariae*: is associated with nephrotic syndrome

Ovale and vivax malaria have a hypnozoite stage and may therefore relapse following treatment.

Treatment

- non-falciparum malarias are almost always chloroquine sensitive
- patients with ovale or vivax malaria should be given primaquine following acute treatment with chloroquine to destroy liver hypnozoites and prevent relapse

External Links

[WHO](#)

Malaria treatment guidelines

Malaria: prophylaxis

There are around 1,500-2,000 cases each year of malaria in patients returning from endemic countries. The majority of these cases (around 75%) are caused by the potentially fatal *Plasmodium falciparum* protozoa. The majority of patients who develop malaria did not take prophylaxis. It should also be remembered that UK citizens who originate from malaria endemic areas quickly lose their innate immunity.

Up-to-date charts with recommended regimes for malarial zones should be consulted prior to prescribing

Drug	Side-effects + notes	Time to begin before travel	Time to end after travel
Atovaquone + proguanil (Malarone)	GI upset	1 - 2 days	7 days
Chloroquine	Headache Contraindicated in epilepsy Taken weekly	1 week	4 weeks
Doxycycline	Photosensitivity Oesophagitis	1 - 2 days	4 weeks
Mefloquine (Lariam)	Dizziness Neuropsychiatric disturbance Contraindicated in epilepsy Taken weekly	2 - 3 weeks	4 weeks
Proguanil (Paludrine)		1 week	4 weeks
Proguanil + chloroquine	See above	1 week	4 weeks

Pregnant women should be advised to avoid travelling to regions where malaria is endemic. Diagnosis can also be difficult as parasites may not be detectable in the blood film due to placental sequestration. However, **if travel cannot be avoided:**

- chloroquine can be taken
- proguanil: folate supplementation (5mg od) should be given
- Malarone (atovaquone + proguanil): the BNF advises to avoid these drugs unless essential. If taken then folate supplementation should be given
- mefloquine: caution advised
- doxycycline is contraindicated.

It is again advisable to avoid travel to malaria endemic regions with children if avoidable. However, if travel is essential then children should take malarial prophylaxis as they are more at risk of serious complications.

- diethyltoluamide (DEET) 20-50% can be used in children over 2 months of age
- doxycycline is only licensed in the UK for children over the age of 12 years

[Clinical Knowledge Summaries](#)

Malaria prophylaxis guidelines

Measles

Overview

- RNA paramyxovirus
- spread by droplets
- infective from prodrome until 4 days after rash starts
- incubation period = 10-14 days

Features

- prodrome: irritable, conjunctivitis, fever
- Koplik spots (before rash): white spots ('grain of salt') on buccal mucosa
- rash: starts behind ears then to whole body, discrete maculopapular rash becoming blotchy & confluent



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Koplik spots

Complications

- encephalitis: typically occurs 1-2 weeks following the onset of the illness)
- subacute sclerosing panencephalitis: very rare, may present 5-10 years following the illness
- febrile convulsions
- giant cell pneumonia
- keratoconjunctivitis, corneal ulceration
- diarrhoea
- increased incidence of appendicitis
- myocarditis



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The rash typically starts behind the ears and then spreads to the whole body

Management of contacts

- if a child not immunized against measles comes into contact with measles then MMR should be offered (vaccine-induced measles antibody develops more rapidly than that following natural infection)
- this should be given within 72 hours

External Links

[Postgraduate Medical Journal](https://www.postgradmedjournal.com/)

Subacute sclerosing panencephalitis

Meningitis: causes

0 - 3 months

- Group B *Streptococcus* (most common cause in neonates)
- *E. coli*
- *Listeria monocytogenes*

3 months - 6 years

- *Neisseria meningitidis*
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*

6 years - 60 years

- *Neisseria meningitidis*
- *Streptococcus pneumoniae*

> 60 years

- *Streptococcus pneumoniae*
- *Neisseria meningitidis*
- *Listeria monocytogenes*

Immunosuppressed

- *Listeria monocytogenes*
-

Meningitis: CSF analysis

The table below summarises the characteristic cerebrospinal fluid (CSF) findings in meningitis:

	Bacterial	Viral	Tuberculous
Appearance	Cloudy	Clear/cloudy	Slight cloudy, fibrin web
Glucose	Low (< 1/2 plasma)	60-80% of plasma glucose*	Low (< 1/2 plasma)
Protein	High (> 1 g/l)	Normal/raised	High (> 1 g/l)
White cells	10 - 5,000 polymorphs/mm ³	15 - 1,000 lymphocytes/mm ³	10 - 1,000 lymphocytes/mm ³

The Ziehl-Neelsen stain is only 20% sensitive in the detection of tuberculous meningitis and therefore PCR is sometimes used (sensitivity = 75%)

*mumps is unusual in being associated with a low glucose level in a proportion of cases. A low glucose may also be seen in herpes encephalitis

External Links

[Medscape](#)

Meningitis: CSF analysis

Meningitis: management

Investigations suggested by NICE

- full blood count
- CRP
- coagulation screen
- blood culture
- whole-blood PCR
- blood glucose
- blood gas

♦Lumbar puncture if no signs of raised intracranial pressure

Management

All patients should be transferred to hospital urgently. If patients are in a pre-hospital setting (for example a GP surgery) and meningococcal disease is suspected then intramuscular benzylpenicillin may be given, as long as this doesn't delay transit to hospital.

BNF recommendations on antibiotics

Scenario	BNF recommendation
Initial empirical therapy aged < 3 months	Intravenous cefotaxime + amoxicillin
Initial empirical therapy aged 3 months - 50 years	Intravenous cefotaxime
Initial empirical therapy aged > 50 years	Intravenous cefotaxime + amoxicillin
Meningococcal meningitis	Intravenous benzylpenicillin or cefotaxime
Pneumococcal meningitis	Intravenous cefotaxime
Meningitis caused by <i>Haemophilus influenzae</i>	Intravenous cefotaxime
Meningitis caused by <i>Listeria</i>	Intravenous amoxicillin + gentamicin

If the patient has a history of immediate hypersensitivity reaction to penicillin or to cephalosporins the BNF recommends using chloramphenicol.

Management of contacts:

- prophylaxis needs to be offered to household and close contacts of patients affected with meningococcal meningitis
- oral ciprofloxacin or rifampicin may be used. The Health Protection Agency (HPA) guidelines now state that whilst either may be used ciprofloxacin is the drug of choice as it is widely available and only requires one dose
- the risk is highest in the first 7 days but persists for at least 4 weeks
- meningococcal vaccination should be offered to close contacts when serotype results are available, including booster doses to those who had the vaccine in infancy
- for pneumococcal meningitis no prophylaxis is generally needed. There are however exceptions to this. If a cluster of cases of pneumococcal meningitis occur the HPA have a protocol for offering close contacts antibiotic prophylaxis. Please see the link for more details

External Links

[Health Protection Agency](#)

2012 Guidance for public health management of meningococcal disease in the UK

[NICE](#)

2010 Bacterial meningitis and meningococcal septicaemia guidelines

[Meningitis Research Foundation](#)

Meningitis protocol

[Health Protection Agency](#)

2008 Guidance on prophylaxis for pneumococcal meningitis

Meningococcal septicaemia: investigations

Meningococcal septicaemia is a frightening condition for patients, parents and doctors. It is associated with a high morbidity and mortality unless treated early - meningococcal disease is the leading infectious cause of death in early childhood. A high index of suspicion is therefore needed. Much of the following is based on the 2010 NICE guidelines (please see link).

Presentation of meningococcal disease:

- 15% - meningitis
- 25% - septicaemia
- 60% - a combination of meningitis and septicaemia

Investigations

- blood cultures
- blood PCR
- lumbar puncture is usually contraindicated
- full blood count and clotting to assess for disseminated intravascular coagulation

External Links

[NICE](#)

2010 Bacterial meningitis and meningococcal septicaemia guidelines

MRSA

Methicillin-resistant *Staphylococcus aureus* (MRSA) was one of the first organisms which highlighted the dangers of hospital-acquired infections.

Who should be screened for MRSA?

- all patients awaiting elective admissions (exceptions include day patients having terminations of pregnancy and ophthalmic surgery. Patients admitted to mental health trusts are also excluded)
- from 2011 all emergency admissions will be screened

How should a patient be screened for MRSA?

- nasal swab and skin lesions or wounds
- the swab should be wiped around the inside rim of a patient's nose for 5 seconds
- the microbiology form must be labelled 'MRSA screen'

Suppression of MRSA from a carrier once identified

- nose: mupirocin 2% in white soft paraffin, tds for 5 days
- skin: chlorhexidine gluconate, od for 5 days. Apply all over but particularly to the axilla, groin and perineum

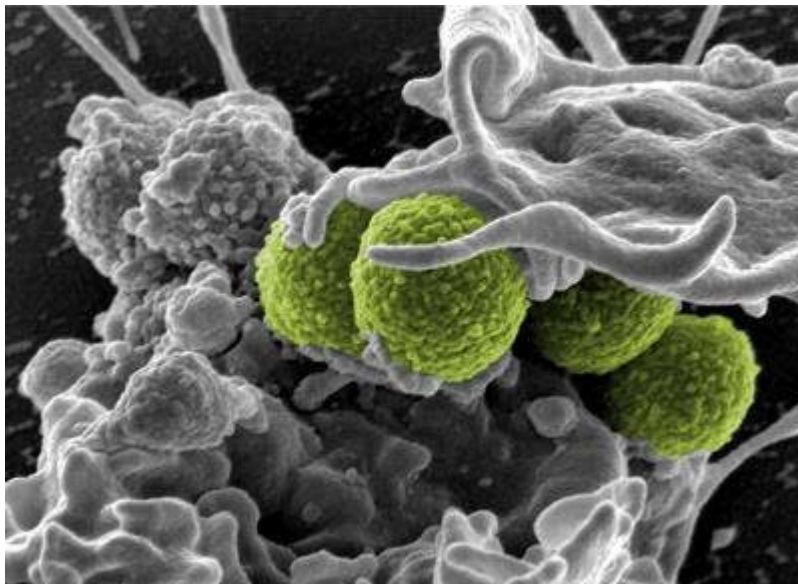
The following antibiotics are commonly used in the treatment of MRSA infections:

- vancomycin
- teicoplanin
- linezolid

Some strains may be sensitive to the antibiotics listed below but they should not generally be used alone because resistance may develop:

- rifampicin
- macrolides
- tetracyclines
- aminoglycosides
- clindamycin

Relatively new antibiotics such as linezolid, quinupristin/dalfopristin combinations and tigecycline have activity against MRSA but should be reserved for resistant cases



Interaction of MRSA (green bacteria) with a human white cell. The bacteria shown is strain MRSA252, a leading cause of hospital-associated infections in the United States and United Kingdom. Credit: NIAID

External Links

[NICE](#)

2012 Infection control guidelines

[Health Protection Agency](#)

2009 MRSA Screening and Suppression guidelines

Necrotising fasciitis

Necrotising fasciitis is a medical emergency that is difficult to recognise in the early stages

It can be classified according to the causative organism:

- type 1 is caused by mixed anaerobes and aerobes (often occurs post-surgery in diabetics)
- type 2 is caused by *Streptococcus pyogenes*

Features

- acute onset
- painful, erythematous lesion develops
- extremely tender over infected tissue

Management

- urgent surgical referral debridement
- intravenous antibiotics

External Links

[DermNet NZ](#)

Necrotising fasciitis

Nematodes

Ancylostoma braziliense:

- most common cause of cutaneous larva migrans
- common in Central and Southern America

Strongyloides stercoralis

- acquired percutaneously (e.g. walking barefoot)
- causes pruritus and larva currens - this has a similar appearance to cutaneous larva migrans but moves through the skin at a far greater rate
- abdo pain, diarrhoea, pneumonitis
- may cause Gram negative septicaemia due carrying of bacteria into bloodstream
- eosinophilia sometimes seen
- management: thiabendazole, albendazole. Ivermectin also used, particularly in chronic infections

Toxocara canis

- commonly acquired by ingesting eggs from soil contaminated by dog faeces
 - commonest cause of visceral larva migrans
 - other features: eye granulomas, liver/lung involvement
-

Orf

Orf is generally a condition found in sheep and goats although it can be transmitted to humans. It is caused by the parapox virus.

In animals

- 'scabby' lesions around the mouth and nose

In humans

- generally affects the hands and arms
 - initially small, raised, red-blue papules
 - later may increase in size to 2-3 cm and become flat-topped and haemorrhagic
-

External Links

[DermNet NZ](#)

Orf

Osteomyelitis

Osteomyelitis describes an infection of the bone.

Staph. aureus is the most common cause except in patients with sickle-cell anaemia where *Salmonella* species predominate.

Predisposing conditions:

- diabetes mellitus
- sickle cell anaemia
- intravenous drug user
- immunosuppression due to either medication or HIV
- alcohol excess

Investigations:

- MRI is the imaging modality of choice, with a sensitivity of 90-100%

Management:

- flucloxacillin for 6 weeks
 - clindamycin if penicillin-allergic
-

Pelvic inflammatory disease

Pelvic inflammatory disease (PID) is a term used to describe infection and inflammation of the female pelvic organs including the uterus, fallopian tubes, ovaries and the surrounding peritoneum. It is usually the result of ascending infection from the endocervix

Causative organisms

- *Chlamydia trachomatis* - the most common cause
- *Neisseria gonorrhoeae*
- *Mycoplasma genitalium*
- *Mycoplasma hominis*

Features

- lower abdominal pain
- fever
- deep dyspareunia
- dysuria and menstrual irregularities may occur
- vaginal or cervical discharge
- cervical excitation

Investigation

- screen for Chlamydia and Gonorrhoea

Management

- due to the difficulty in making an accurate diagnosis, and the potential complications of untreated PID, consensus guidelines recommend having a low threshold for treatment
- oral ofloxacin + oral metronidazole or intramuscular ceftriaxone + oral doxycycline + oral metronidazole
- RCOG guidelines suggest that in mild cases of PID intrauterine contraceptive devices may be left in. The more recent BASHH guidelines suggest that the evidence is limited but that '*Removal of the IUD should be considered and may be associated with better short term clinical outcomes*'.

Complications

- infertility - the risk may be as high as 10-20% after a single episode
- chronic pelvic pain
- ectopic pregnancy

External Links

[British Association for Sexual Health and HIV \(BASHH\)](#)

UK National Guideline for the Management of Pelvic Inflammatory Disease 2011

Post-exposure prophylaxis

Hepatitis A

- Human Normal Immunoglobulin (HNIG) or hepatitis A vaccine may be used depending on the clinical situation

Hepatitis B

- HBsAg positive source: if the person exposed is a known responder to HBV vaccine then a booster dose should be given. If they are in the process of being vaccinated or are a non-responder they need to have hepatitis B immune globulin (HBIG) and the vaccine
- unknown source: for known responders the green book advises considering a booster dose of HBV vaccine. For known non-responders HBIG + vaccine should be given whilst those in the process of being vaccinated should have an accelerated course of HBV vaccine

Hepatitis C

- monthly PCR - if seroconversion then interferon +/- ribavirin

HIV

- a combination of oral antiretrovirals (e.g. Tenofovir, emtricitabine, lopinavir and ritonavir) as soon as possible (i.e. Within 1-2 hours, but may be started up to 72 hours following exposure) for 4 weeks
- serological testing at 12 weeks following completion of post-exposure prophylaxis
- reduces risk of transmission by 80%

Varicella zoster

- VZIG for IgG negative pregnant women/immunosuppressed

Estimates of transmission risk for single needlestick injury

Hepatitis B	20-30%
Hepatitis C	0.5-2%
HIV	0.3%

External Links

[Greenbook](#)

Hepatitis B

[BHIVA](#)

Post-exposure prophylaxis following sexual exposure

[Department of health](#)

HIV post-exposure prophylaxis

Pyrexia of unknown origin

Defined as a prolonged fever of > 3 weeks which resists diagnosis after a week in hospital

Neoplasia

- lymphoma
- hypernephroma
- preleukaemia
- atrial myxoma

Infections

- abscess
- TB

Connective tissue disorders

Rabies

Rabies is a viral disease that causes an acute encephalitis. The rabies virus is classed as a RNA rhabdovirus and has a bullet shaped capsid. It is commonly transmitted by bat, raccoon and skunk bites. Following a bite the virus travels up the nerve axons towards the central nervous system in a retrograde fashion.

Features

- prodrome: headache, fever, agitation
- hydrophobia: water-provoking muscle spasms
- hypersalivation
- Negri bodies: cytoplasmic inclusion bodies found in infected neurons

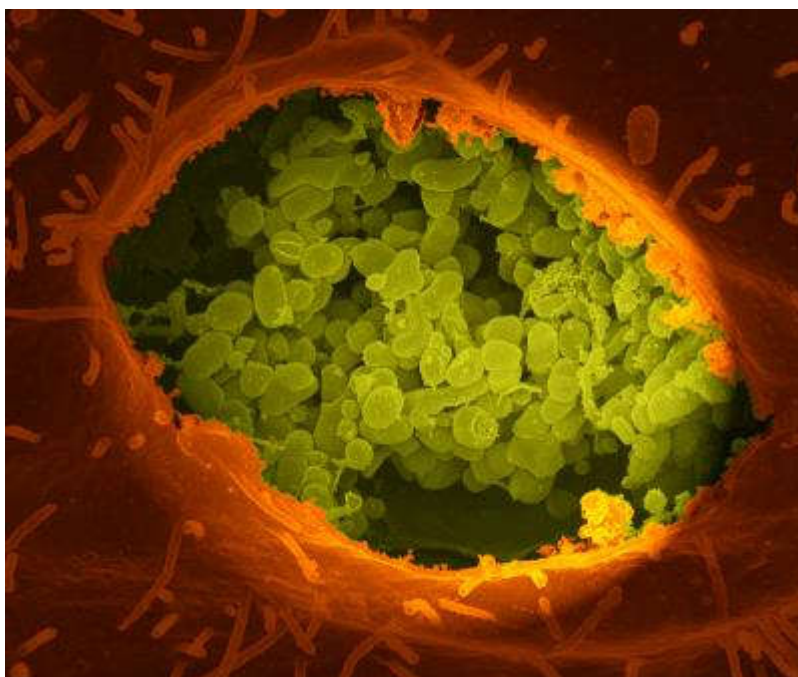
There is now considered to be 'no risk' of developing rabies following an animal bite in the UK and the majority of developed countries. **Following an animal bite in at risk countries:**

- if an individual is already immunised then 2 further doses of vaccine should be given
- if not previously immunised then human rabies immunoglobulin (HRIG) should be given along with a full course of vaccination

Rickettsiae

Rickettsiae are Gram-negative obligate intracellular parasites. Types of rickettsiae cause a variety of diseases that are typically characterised by fever, headache and rash. A notable exception is Q fever (cause by *Coxiella burnetti* which causes pneumonia but no rash. The Weil-Felix reaction is positive except in Q fever. Rickettsial diseases are all treated with tetracyclines.

Disease	Cause	Vector	Notes
Rocky Mountain spotted fever	<i>Rickettsia rickettsii</i>	Tick	Headache and fever are common Rash starts on the peripheries (wrist, ankles) before spreading centrally. It is initially maculopapular before becoming vasculitic Endemic to east coast of US
Q fever	<i>Coxiella burnetti</i>	No vector	No rash but causes pneumonia
Endemic typhus	<i>Rickettsia typhi</i>	Flea	Rash starts centrally then spreads to the peripheries
Epidemic typhus	<i>Rickettsia prowazekii</i>	Human body louse	
Ehrlichiosis	<i>Ehrlichia</i>	Tick	



A dry fracture of a Vero cell exposing the contents of a vacuole where *Coxiella burnetii* are busy growing. Note the intracellular nature of the organism. Credit: NIAID

RNA viruses

Family	Structure	Envelope	Capsid Symmetry	Examples
Reoviridae	Double-stranded , linear	Naked	Icosahedral	Reovirus, Rotavirus
Picornaviridae	Single-stranded +, linear	Naked	Icosahedral	Enterovirus, Rhinovirus, Poliovirus, Cocksackie
Caliciviridae	Single-stranded +, linear	Naked	Icosahedral	Norwalk virus
Togaviridae	Single-stranded +, linear	Enveloped	Icosahedral	Rubella virus
Arenaviridae	Single-stranded -, circular	Enveloped	Helical	Lymphocytic choriomeningitis virus
Flaviviridae	Single-stranded +, linear	Enveloped	Icosahedral	Dengue virus, Hepatitis C virus, Yellow fever virus
Orthomyxoviridae	Single-stranded -, linear	Enveloped	Helical	Influenzavirus A, Influenzavirus B, Influenzavirus C,
Paramyxoviridae	Single-stranded -, linear	Enveloped	Helical	Measles virus, Mumps virus, Respiratory syncytial virus
Bunyaviridae	Single-stranded -, circular	Enveloped	Helical	California encephalitis virus, Hantavirus
Rhabdoviridae	Single-stranded -, linear	Enveloped	Helical	Rabies virus
Filoviridae	Single-stranded -, linear	Enveloped	Helical	Ebola virus, Marburg virus
Coronaviridae	Single-stranded +, linear	Enveloped	Helical	Corona virus
Hepeviridae	Single-stranded +, linear	Naked	Icosahedral	Hepatitis E virus

RNA viruses memory aids:

- Reoviridae are the only double-stranded RNA viruses
- 'CALL PICO and FLAVA. There's a RETRO TOGA party with lots of CORONAs'

Salmonella

♦The *Salmonella* group contains many members, most of which cause diarrhoeal diseases. They are aerobic, Gram negative rods which are not normally present as commensals in the gut.

♦Typhoid and paratyphoid are caused by *Salmonella typhi* and *Salmonella paratyphi* (types A, B & C) respectively. They are often termed enteric fevers, producing systemic symptoms such as headache, fever, arthralgia

Features

- initially systemic upset as above
- relative bradycardia
- abdominal pain, distension
- constipation: although *Salmonella* is a recognised cause of diarrhoea, constipation is more common in typhoid
- rose spots: present on the trunk in 40% of patients, and are more common in paratyphoid

Possible complications include:

- osteomyelitis (especially in sickle cell disease where *Salmonella* is one of the most common pathogens)
- GI bleed/perforation
- meningitis
- cholecystitis
- chronic carriage (1%, more likely if adult females)

External Links

[DermNet NZ](#)

Typhoid fever review

Scabies

Scabies is caused by the mite *Sarcoptes scabiei* and is spread by prolonged skin contact. It typically affects children and young adults.

The scabies mite burrows into the skin, laying its eggs in the stratum corneum. The intense pruritus associated with scabies is due to a delayed type IV hypersensitivity reaction to mites/eggs which occurs about 30 days after the initial infection.

Features

- widespread pruritus
- linear burrows on the side of fingers, interdigital webs and flexor aspects of the wrist
- in infants the face and scalp may also be affected
- secondary features are seen due to scratching: excoriation, infection

Management

- permethrin 5% is first-line
- malathion 0.5% is second-line
- give appropriate guidance on use (see below)
- pruritus persists for up to 4-6 weeks post eradication

Patient guidance on treatment (from Clinical Knowledge Summaries):

- avoid close physical contact with others until treatment is complete
- all household and close physical contacts should be treated at the same time, even if asymptomatic
- launder, iron or tumble dry clothing, bedding, towels, etc., on the first day of treatment to kill off mites.

The BNF advises to apply the insecticide to all areas, including the face and scalp, contrary to the manufacturer's recommendation. **Patients should be given the following instructions:**

- apply the insecticide cream or liquid to cool, dry skin
- pay close attention to areas between fingers and toes, under nails, armpit area, creases of the skin such as at the wrist and elbow
- allow to dry and leave on the skin for 8-12 hours for permethrin, or for 24 hours for malathion, before washing off
- reapply if insecticide is removed during the treatment period, e.g. If wash hands, change nappy, etc
- repeat treatment 7 days later

Crusted (Norwegian) scabies

Crusted scabies is seen in patients with suppressed immunity, especially HIV.



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The crusted skin will be teeming with hundreds of thousands of organisms.

Ivermectin is the treatment of choice and isolation is essential

External Links

[Clinical Knowledge Summaries](#)

Scabies guidelines

Schistosomiasis

Schistosomiasis, or bilharzia, is a parasitic flatworm infection. **The following types of schistosomiasis are recognised:**

- Schistosoma mansoni and Schistosoma intercalatum: intestinal schistosomiasis
- Schistosoma haematobium: urinary schistosomiasis

Schistosoma haematobium

This typically presents as a 'swimmer's itch' in patients who have recently returned from Africa. Schistosoma haematobium is a risk factor for squamous cell bladder cancer

Features

- frequency
- haematuria
- bladder calcification

Management

- single oral dose of praziquantel

External Links

[Royal College of Physicians](#)

2011 Schistosomiasis review

Shigella

Overview

- causes bloody diarrhoea, abdo pain
- severity depends on type: S sonnei (e.g. from UK) may be mild, S flexneri or S dysenteriae from abroad may cause severe disease
- treat with ciprofloxacin

Splenectomy

Following a splenectomy patients are particularly at risk from pneumococcus, Haemophilus, meningococcus and Capnocytophaga canimorsus* infections

Vaccination

- if elective, should be done 2 weeks prior to operation
- Hib, meningitis A & C
- annual influenza vaccination
- pneumococcal vaccine every 5 years

Antibiotic prophylaxis

- penicillin V: unfortunately clear guidelines do not exist of how long antibiotic prophylaxis should be continued. It is generally accepted though that penicillin should be continued for at least 2 years and at least until the patient is 16 years of age, although the majority of patients are usually put on antibiotic prophylaxis for life

Surgical aspects

Indications

- Trauma: 1/4 are iatrogenic
- Spontaneous rupture: EBV
- Hypersplenism: hereditary spherocytosis or elliptocytosis etc
- Malignancy: lymphoma or leukaemia
- Splenic cysts, hydatid cysts, splenic abscesses

Splenectomy following trauma:

- GA
- Long midline incision
- If time permits insert a self retaining retractor (e.g. Balfour/ omnitract)
- Large amount of free blood is usually present. Pack all 4 quadrants of the abdomen. Allow the anaesthetist to 'catch up'
- Remove the packs and assess the viability of the spleen. Hilar injuries and extensive parenchymal lacerations will usually require splenectomy.
- Divide the short gastric vessels and ligate them.
- Clamp the splenic artery and vein. Two clamps on the patient side are better and allow for double ligation and serve as a safety net if your assistant does not release the clamp smoothly.
- Be careful not to damage the tail of the pancreas, if you do then this will need to be formally removed and the pancreatic duct closed.
- Wash out the abdomen and place a tube drain to the splenic bed.
- Some surgeons implant a portion of spleen into the omentum, whether you decide to do this is a matter of personal choice.
- Postoperatively the patient will require prophylactic penicillin V and pneumococcal vaccine.

Elective splenectomy

- Elective splenectomy is a very different operation from that performed in the emergency setting. The spleen is often large (sometimes massive)
- Most cases can be performed laparoscopically. The spleen will often be macerated inside a specimen bag to facilitate extraction.

Complications

- Haemorrhage (may be early and either from short gastrics or splenic hilar vessels)
- Pancreatic fistula (from iatrogenic damage to pancreatic tail)
- Thrombocytosis: prophylactic aspirin
- Encapsulated bacteria infection e.g. *Strep. pneumoniae*, *Haemophilus influenzae* and *Neisseria meningitidis*

Post-splenectomy changes:

- Platelets will rise first (therefore in ITP should be given after splenic artery clamped)
- Blood film will change over following weeks, Howell-Jolly bodies will appear
- Other blood film changes include target cells and Pappenheimer bodies
- Increased risk of post-splenectomy sepsis, therefore prophylactic antibiotics and pneumococcal vaccine should be given.

Post-splenectomy sepsis

- Typically occurs with encapsulated organisms
- Opsonisation occurs but then not recognised

*usually from dog bites

External Links

[Patient.info](#)

Splenectomy and immunization review

Staphylococcal toxic shock syndrome

Staphylococcal toxic shock syndrome describes a severe systemic reaction to staphylococcal exotoxins. It came to prominence in the early 1980's following a series of cases related to infected tampons

Centers for Disease Control and Prevention diagnostic criteria

- fever: temperature $> 38.9^{\circ}\text{C}$
- hypotension: systolic blood pressure < 90 mmHg
- diffuse erythematous rash
- desquamation of rash, especially of the palms and soles
- involvement of three or more organ systems: e.g. gastrointestinal (diarrhoea and vomiting), mucous membrane erythema, renal failure, hepatitis, thrombocytopenia, CNS involvement (e.g. confusion)



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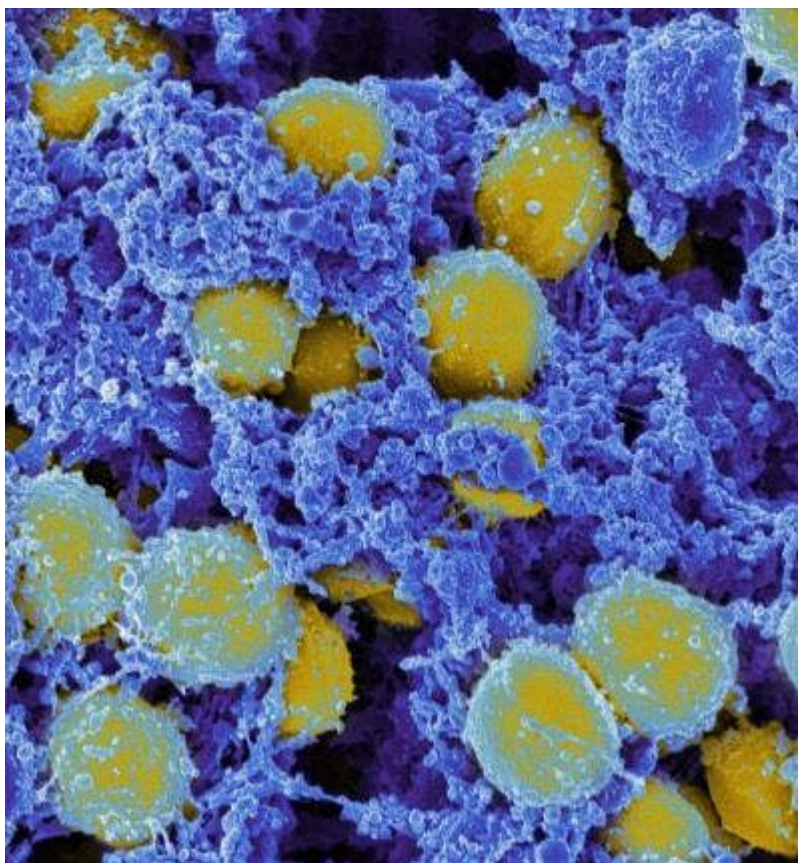
Staphylococci

Staphylococci are a common type of bacteria which are often found normal commensal organisms but may also cause invasive disease. **Some basic facts include:**

- Gram-positive cocci
- facultative anaerobes
- produce catalase

The two main types of Staphylococci you need to know about are *Staphylococcus aureus* and *Staphylococcus epidermidis*.

<i>Staphylococcus aureus</i>	<i>Staphylococcus epidermidis</i>
• Coagulase-positive • Causes skin infections (e.g. cellulitis), abscesses, osteomyelitis, toxic shock syndrome	• Coagulase-negative • Cause of central line infections and infective endocarditis



Scanning electromicrograph of Staphylococcus aureus bacteria. Credit: NIAID

STI: ulcers

◆Genital herpes is most often caused by the herpes simplex virus (HSV) type 2 (cold sores are usually due to HSV type 1). Primary attacks are often severe and associated with fever whilst subsequent attacks are generally less severe and localised to one site

◆Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. A painless ulcer (chancre) is seen in the primary stage. The incubation period= 9-90 days

◆Chancroid is a tropical disease caused by *Haemophilus ducreyi*. It causes painful genital ulcers associated with unilateral, painful inguinal lymph node enlargement. The ulcers typically have a sharply defined, ragged, undermined border.

Chapter: Infectious Diseases & STI

Lymphogranuloma venereum (LGV) is caused by *Chlamydia trachomatis*. **Typically infection comprises of three stages**

- **stage 1:** small painless pustule which later forms an ulcer
- **stage 2:** painful inguinal lymphadenopathy
- **stage 3:** proctocolitis

LGV is treated using doxycycline.

Other causes of genital ulcers

- Behcet's disease
- carcinoma
- granuloma inguinale: *Klebsiella granulomatis**

*previously called *Calymmatobacterium granulomatis*

External Links

[British Association for Sexual Health and HIV](#)

2011 Guideline for the management of Donovanosis (Granuloma inguinale)

Streptococci

Streptococci are gram-positive cocci. They may be divided into alpha and beta haemolytic types

Alpha haemolytic streptococci (partial haemolysis)

The most important alpha haemolytic *Streptococcus* is *Streptococcus pneumoniae* (pneumococcus). Pneumococcus is a common cause of pneumonia, meningitis and otitis media. Another clinical example is *Streptococcus viridans*

Beta haemolytic streptococci (complete haemolysis)

These can be subdivided into groups A-H. Only groups A, B & D are important in humans.

Group A

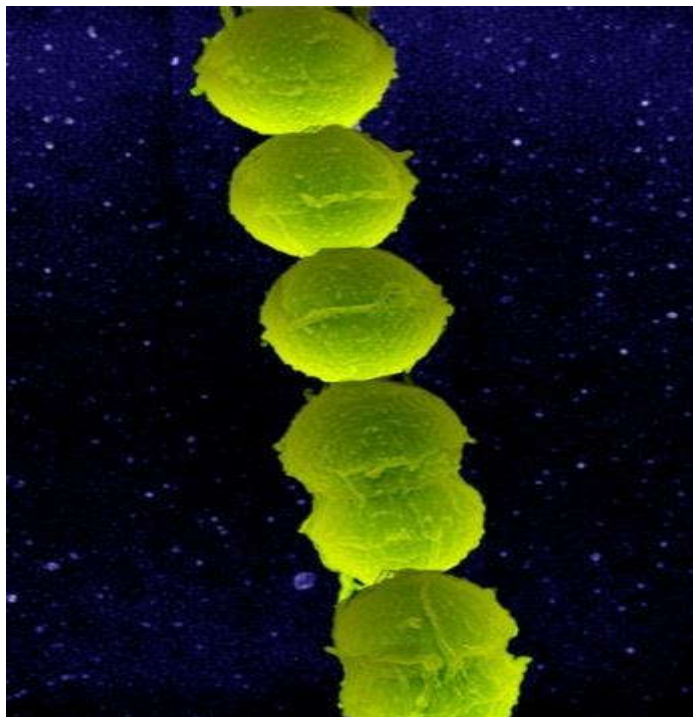
- most important organism is *Streptococcus pyogenes*
- responsible for erysipelas, impetigo, cellulitis, type 2 necrotizing fasciitis and pharyngitis/tonsillitis
- immunological reactions can cause rheumatic fever or post-streptococcal glomerulonephritis
- erythrogenic toxins cause scarlet fever

Group B

- *Streptococcus agalactiae* may lead to neonatal meningitis and septicaemia

Group D

- *Enterococcus*



Group B streptococcus bacteria. Credit: NIAID

External Links

[DermNet NZ](#)

Erysipelas

Strongyloides stercoralis

Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with *Strongyloides stercoralis* causes strongyloidiasis.

Features

- diarrhoea
- abdominal pain/bloating
- papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks
- larva currens: pruritic, linear, urticarial rash
- if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered

Treatment

- ivermectin and albendazole are used

Syphilis

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. The incubation period is between 9-90 days

Primary features

- chancre - painless ulcer at the site of sexual contact
- local non-tender lymphadenopathy
- often not seen in women (the lesion may be on the cervix)

Secondary features - occurs 6-10 weeks after primary infection

- systemic symptoms: fevers, lymphadenopathy
- rash on trunk, palms and soles
- buccal 'snail track' ulcers (30%)
- condylomata lata



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Classical palm lesions of secondary syphilis



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More generalised rash of secondary syphilis

Tertiary features

- gummas (granulomatous lesions of the skin and bones)
- ascending aortic aneurysms
- general paralysis of the insane
- tabes dorsalis
- Argyll-Robertson pupil

Features of congenital syphilis

- blunted upper incisor teeth (Hutchinson's teeth), 'mulberry' molars
- rhagades (linear scars at the angle of the mouth)
- keratitis
- saber shins
- saddle nose
- deafness

External Links

[Clinical Knowledge Summaries](#)

Syphilis guidelines

[DermNet NZ](#)

Syphilis images

[DermIS.net](#)

Picture of condylomata lata

Syphilis: investigation

Treponema pallidum is a very sensitive organism and cannot be grown on artificial media. The diagnosis is therefore usually based on clinical features, serology and microscopic examination of infected tissue

Serological tests can be divided into:

- cardiolipin tests (not treponeme specific)
- treponemal specific antibody tests

Cardiolipin tests

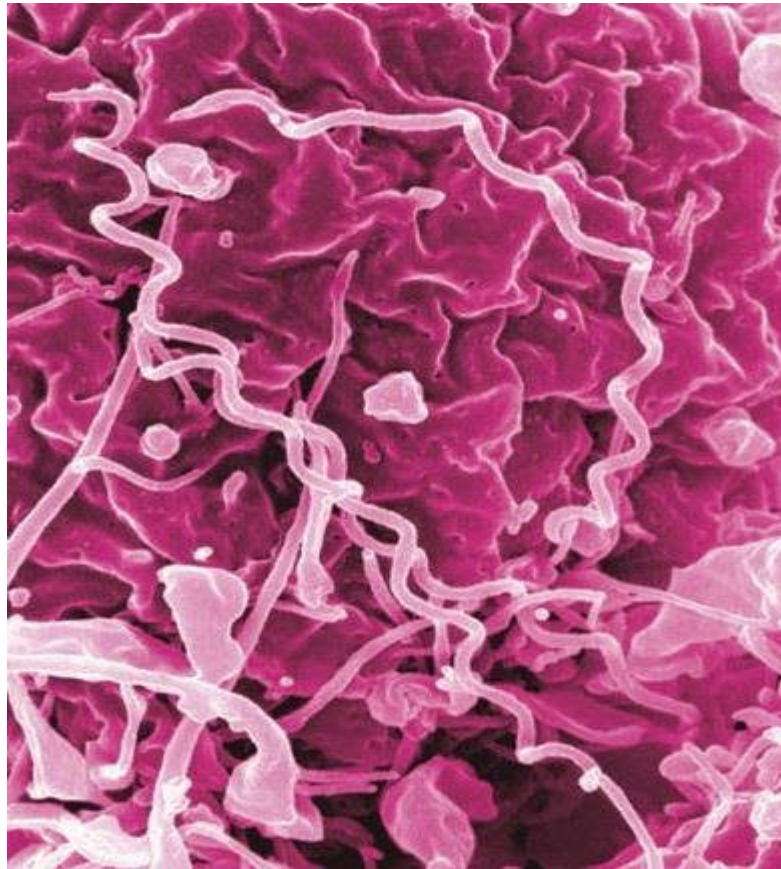
- syphilis infection leads to the production of non-specific antibodies that react to cardiolipin
- examples include VDRL (Venereal Disease Research Laboratory) & RPR (rapid plasma reagin)
- insensitive in late syphilis
- becomes negative after treatment

Treponemal specific antibody tests

- example: TPHA (*Treponema pallidum* HaemAgglutination test)
- remains positive after treatment

Causes of false positive cardiolipin tests

- pregnancy
- SLE, anti-phospholipid syndrome
- TB
- leprosy
- malaria
- HIV



Treponema pallidum, the bacteria that cause syphilis. Note the spiral shape of the organism.
Credit: NIAID

Syphilis: management

Management

- benzylpenicillin
- alternatives: doxycycline
- the Jarisch-Herxheimer reaction is sometimes seen following treatment. Fever, rash, tachycardia after first dose of antibiotic. It is thought to be due to the release of endotoxins following bacterial death and typically occurs within a few hours of treatment.

External Links

[British Association for Sexual Health and HIV](#)

UK National Guidelines on the Management of Syphilis 2015

Tape worms

Tape worms are made up of repeated segments called proglottids. These are often present in faeces and are useful diagnostically

Cysticercosis

- caused by *Taenia solium* (from pork) and *Taenia saginata* (from beef)
- management: niclosamide

Hydatid disease

- caused by the dog tapeworm *Echinococcus granulosus*
- life-cycle involves dogs ingesting hydatid cysts from sheep liver
- often seen in farmers
- may cause liver cysts
- management: albendazole

Tetanus

Tetanus is caused by the tetanospasmin exotoxin released from *Clostridium tetani*. Tetanus spores are present in soil and may be introduced into the body from a wound, which is often unnoticed. Tetanospasmin prevents release of GABA

Features

- prodrome fever, lethargy, headache
- trismus (lockjaw)
- risus sardonicus
- opisthotonus (arched back, hyperextended neck)
- spasms (e.g. dysphagia)

Management

- supportive therapy including ventilatory support and muscle relaxants
- intramuscular human tetanus immunoglobulin for high-risk wounds (e.g. compound fractures, delayed surgical intervention, significant degree of devitalised tissue)
- metronidazole is now preferred to benzylpenicillin as the antibiotic of choice

Tetanus: vaccination

The tetanus vaccine is a cell-free purified toxin that is normally given as part of a combined vaccine.

Tetanus vaccine is currently given in the UK as part of the routine immunisation schedule at:

- 2 months
- 3 months
- 4 months
- 3-5 years
- 13-18 years

This therefore provides 5 doses of tetanus-containing vaccine. Five doses is now considered to provide adequate long-term protection against tetanus.

Intramuscular human tetanus immunoglobulin should be given to patients with high-risk wounds (e.g. Compound fractures, delayed surgical intervention, significant degree of devitalised tissue) irrespective of whether 5 doses of tetanus vaccine have previously been given

If vaccination history is incomplete or unknown then a dose of tetanus vaccine should be given combined with intramuscular human tetanus immunoglobulin for high-risk wounds

External Link

[Department of Health](#)

Tetanus guidelines

Toxoplasmosis

◆ *Toxoplasma gondii* is a protozoa which infects the body via the GI tract, lung or broken skin. Its oocysts release trophozoites which migrate widely around the body including to the eye, brain and muscle. The usual animal reservoir is the cat, although other animals such as rats carry the disease.

◆ Most infections are asymptomatic. Symptomatic patients usually have a self-limiting infection, often having clinical features resembling infectious mononucleosis (fever, malaise, lymphadenopathy). Other less common manifestations include meningoencephalitis and myocarditis.

Investigation

- antibody test
- Sabin-Feldman dye test

Treatment is usually reserved for those with severe infections or patients who are immunosuppressed

- pyrimethamine plus sulphadiazine for at least 6 weeks

Congenital toxoplasmosis is due to transplacental spread from the mother. It causes a variety of effects to the unborn child including microcephaly, hydrocephalus, cerebral calcification and choroidoretinitis.

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease)

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. **Clinical features include:**

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atrophy) and arrhythmias
- gastrointestinal features includes megaesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

External Links

[DermNet NZ](#)

Chaga's disease

Tuberculosis: diagnosis

In adults induction of sputum or bronchoscopy and lavage may be used in patients who cannot produce sputum

In children who are unable to cough up sputum, the gold standard is gastric washings for M tuberculosis culture

Urinary tract infection in adults: management

Lower urinary tract infections

Non-pregnant women

- local antibiotic guidelines should be followed if available
- 2012 SIGN guidelines recommend trimethoprim or nitrofurantoin for 3 days

Pregnant women with symptomatic bacteriuria should be treated with an antibiotic for 7 days. A urine culture should be sent. **For asymptomatic pregnant women:**

- a urine culture should be performed routinely at the first antenatal visit
- if positive, a second urine culture should be sent to confirm the presence of bacteriuria
- SIGN recommend to treat asymptomatic bacteriuria detected during pregnancy with an antibiotic
- a 7 day course of antibiotics should be given
- a further urine culture should be sent following completion of treatment as a test of cure

Acute pyelonephritis

For patients with sign of acute pyelonephritis hospital admission should be considered:

- local antibiotic guidelines should be followed if available
- the BNF currently recommends a broad-spectrum cephalosporin or a quinolone (for non-pregnant women) for 10-14 days

External Links

[SIGN](#)

2012 Management of suspected bacterial urinary tract infection in adults

[Clinical Knowledge Summaries](#)

Management of UTI in pregnant women

Vaccinations

It is important to be aware of vaccines which are of the live-attenuated type as these may pose a risk to immunocompromised patients. The main types of vaccine are as follows:

Live attenuated

- BCG
- measles, mumps, rubella (MMR)
- influenza (intranasal)
- oral rotavirus
- oral polio
- yellow fever
- oral typhoid*

Inactivated preparations

- rabies
- influenza (intramuscular)

Detoxified exotoxins

- tetanus

Extracts of the organism/virus (sometimes termed fragment)**

- diphtheria
- pertussis ('acellular' vaccine)
- hepatitis B
- meningococcus, pneumococcus, haemophilus

Notes:

- influenza: different types are available, including whole inactivated virus, split virion (virus particles disrupted by detergent treatment) and sub-unit (mainly haemagglutinin and neuraminidase)
- cholera: contains inactivated Inaba and Ogawa strains of *Vibrio cholerae* together with recombinant B-subunit of the cholera toxin
- hepatitis B: contains HBsAg adsorbed onto aluminium hydroxide adjuvant and is prepared from yeast cells using recombinant DNA technology

*whole cell typhoid vaccine is no longer used in the UK

**may also be produced using recombinant DNA technology

Vaginal discharge

Vaginal discharge is a common presenting symptom and is not always pathological

Common causes

- physiological
- *Candida*
- *Trichomonas vaginalis*
- bacterial vaginosis

Less common causes

- *Gonorrhoea*
- *Chlamydia* can cause a vaginal discharge although this is rarely the presenting symptoms
- ectropion
- foreign body
- cervical cancer

Key features of the common causes are listed below

Condition	Key features
<i>Candida</i>	'Cottage cheese' discharge Vulvitis Itch
<i>Trichomonas vaginalis</i>	Offensive, yellow/green, frothy discharge Vulvovaginitis Strawberry cervix
Bacterial vaginosis	Offensive, thin, white/grey, 'fishy' discharge

External Links

[British Association for Sexual Health & HIV \(BASHH\)](#)

Guidelines for a variety of sexual health related problems

Vancomycin

Vancomycin is a glycopeptide antibiotic used in the treatment of Gram positive infections, particularly methicillin-resistant *Staphylococcus aureus* (MRSA).

Mechanism of action

- inhibits cell wall formation by binding to D-Ala-D-Ala moieties, preventing polymerization of peptidoglycans

Mechanism of resistance

- alteration to the terminal amino acid residues of the NAM/NAG-peptide subunits (normally D-alanyl-D-alanine) to which the antibiotic binds

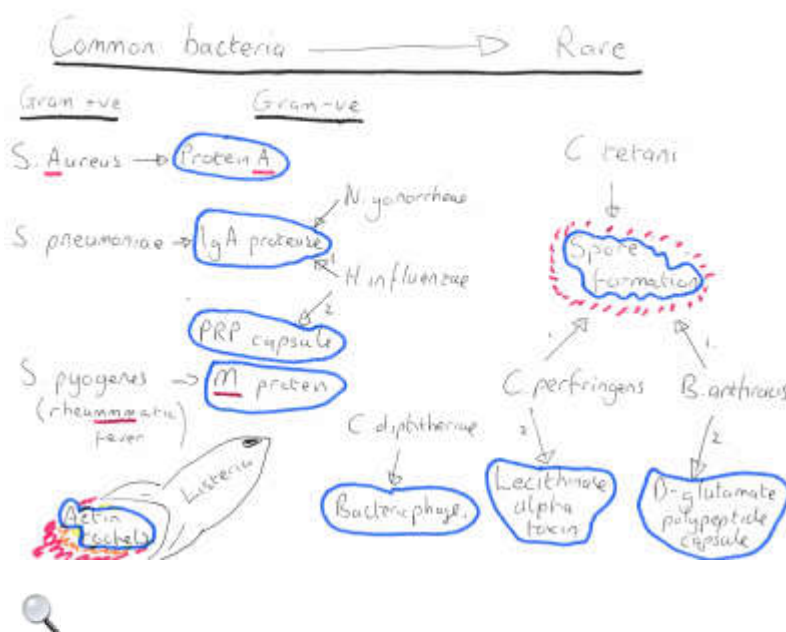
Adverse effects

- nephrotoxicity
- ototoxicity
- thrombophlebitis
- red man syndrome; occurs on rapid infusion of vancomycin

Virulence factors

Bacteria employ a large number of virulence factors which enable them to colonize the host and evade/suppress the immune response. The table below shows a select number of virulence factors which are important for the exam.

Virulence factor	Example organisms
IgA protease	<i>Streptococcus pneumoniae</i> <i>Haemophilus influenzae</i> <i>Neisseria gonorrhoeae</i>
M Protein	<i>Streptococcus pyogenes</i>
Polyribosyl ribitol phosphate capsule	<i>Haemophilus influenzae</i>
Bacteriophage	<i>Corynebacterium diphtheriae</i>
Spore formation	<i>Bacillus anthracis</i> <i>Clostridium perfringens</i> <i>Clostridium tetani</i>
Lecithinase alpha toxin	<i>Clostridium perfringens</i>
D-glutamate polypeptide capsule	<i>Bacillus anthracis</i>
Actin rockets	<i>Listeria monocytogenes</i>



Zika virus

Zika is a mosquito-borne infection caused by Zika virus, a member of the genus flavivirus and family Flaviviridae. It was first isolated from a monkey in the Zika forest in Uganda in 1947.

Transmission is usually via the bite of an infected Aedes mosquito, although a small number of cases of sexual transmission have been reported. There is increasing evidence of transmission via the placenta from mother to fetus.

The majority of people infected with Zika virus have no symptoms. For those with symptoms, Zika virus tends to cause a mild, short-lived (2 to 7 days) febrile disease. **Signs and symptoms suggestive of Zika virus infection may include a combination of the following:**

- fever
- rash
- arthralgia/arthritis
- conjunctivitis
- myalgia
- headache
- retro-orbital pain
- pruritus

Serious complications in adults are not common, although the virus has been associated with Guillain-Barre syndrome. Scientific consensus however has linked Zika with microcephaly and other congenital abnormalities, which has led the World Health Organisation (WHO) to declare a Public Health Emergency of International Concern (PHEIC).

Advice for travellers

There is currently no vaccine or drug to prevent Zika infection. Prevention revolves around avoiding mosquito bites (Aedes mosquitoes usually bite during the day) by using mosquito repellent and cover up clothing. Pregnant women are advised to avoid non-essential travel to Zika prevalent areas until after pregnancy.

External Links

[Public Health England](#)

Zika virus guidance